THE AMERICAN JOURNAL OF ROENTGENOLOGY AND RADIUM THERAPY

VOL. 51 JUNE, 1944 No. 6

ACTINOMYCOSIS OF THE VERTEBRAE

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ACTINOMYCOSIS of the vertebrae is a rare manifestation of a relatively rare disease. Yet this unusual complication presents such a characteristic roentgen appearance when it does occur that it is strange to find little mention of it in roentgenologic textbooks, and only scattered mention of it in roentgenologic periodicals, the latter consisting of brief summaries of articles originally presented elsewhere.

In the past year, 2 cases of actinomycosis with vertebral involvement have occurred at City Hospital and a less recent case has been found at the University Hospitals of Cleveland, all of which present lesions well demonstrated on the roentgenogram. It is the purpose of this paper to report these 3 cases with postmortem as well as roentgen findings.

INCIDENCE

In 1923 Sanford[11] published a series of 678 cases of actinomycosis, only 4 of which had rib or vertebral involvement. At approximately the same time Parker[10] presented 9 cases of vertebral actinomycosis which he collected from the literature. By 1927 Simpson and McIntosh[13] were able to collect from the world literature 28 cases of vertebral actinomycosis, 13 of which were in the American literature. To this group they added 4 of their own cases. Snoke[14] added a case in 1928 and Ward[17] another in 1931. In 1933 Tabb and Tucker[15] again reviewed the subject and added another case report. Their presentation has the only illustrations of the roentgen manifestations of the disease in recent American roentgenologic periodicals. In 1935 Meyer and Gall[8] reviewed the literature completely and collected 47 cases of vertebral actinomycosis. They present an excellent study of the subject, but without illustrative roentgenograms. Other articles presenting single cases have occurred subsequently; the one presented by Dixon[4] resembles one of the cases presented here in that collapse of a vertebra occurred. This is a rare phenomenon. Since many of the cases are usually called Pott's disease before autopsy it may be that with greater awareness of its existence the diagnosis of vertebral actinomycosis will be made more frequently.

ETIOLOGY

The precise etiology of actinomycosis has been the subject of much debate, but in recent years most of the argument has been resolved mainly by the researches of Naeslund[9] and the popularization by Cope.[1,2] It was generally agreed that actinomyces caused the disease. However, the pathogenicity of the various varieties, the cultural characteristics of the organisms re-

covered in human lesions, the life cycle of the various types with the possibility of one type reverting to another in a different phase of development, and even the nomenclature were at times parts of an active controversy.

The present conception of actinomycosis is that it is caused in a great preponderance of cases by an anaerobic organism which normally inhabits many human mouths. On rare occasions an aerobic organism which has its origin outside the body may be responsible. The organisms found on grasses and grains are aerobic, but are not believed to be pathogenic. The historical development of these concepts is interestingly presented by Cope.[1]

These beliefs are presented for more than academic interest. They are important for several reasons: first, the victim of the disease need not be a farm worker or one who has had contact with cattle; second, he need not have chewed grasses, straws, or other vegetable matter; and third, such incidents in the patient's history as a recent tonsillectomy, poor mouth hygiene, a recent tooth extraction or a human bite are as much or even more important in suggesting actinomycosis than the occupational and social history. Many recent articles[2,3,7] support these views. Nevertheless their appreciation is not widespread.

At the present time there has been a general tendency to abandon all the confusion in terminology with the acceptance of the general term, "actinomyces," for the entire group of organisms and "actinomycosis" for the disease. The term, "streptothrix," has largely been dropped except as a subtitle. However Lord[7] still believes it to be a useful designation for the rare aerobic form. He believes morphological and prognostic differences substantiate his point. Cope, on the other hand, is not impressed and believes unity in nomenclature is more valuable.

PATHOGENESIS AND PATHOLOGIC FINDINGS

The vertebrae are involved secondarily usually by direct extension but occasionally by blood stream metastasis. The organism sets up the primary focus by passing through the mucous membranes of some portion of the respiratory or gastrointestinal tract. Occasionally entrance may be made through the skin. Suppuration begins and its main feature is extensive burrowing with little respect for anatomical barriers. Thus the vertebrae can be reached from almost any primary site, but mainly from pulmonary and gastrointestinal foci.

When the infection reaches the vertebrae it is usually in the form of a paravertebral phlegmon and in the abdomen may resemble a tuberculous psoas abscess. The cortex of the vertebrae is first eroded and abscesses are set up beneath it. Osseous condensation takes place surrounding the abscesses at the center of which the organisms may be found. While all portions of the vertebra may be involved, including the processes and lamina as well as the bodies, the intervertebral discs are usually spared and angular deformity is rare.

Pathologically, the disease belongs to the group of chronic granulomas. However, there are several interesting features peculiar to it. In the early stages extreme vascularity is one of them. During surgical operation, bleeding is profuse and the alert surgeon[13,16] may suspect the presence of the disease at the time of operation because of it. When actinomycosis has been present for a longer period of time, much glistening, firm, connective tissue is laid down in the form of thick strands which surround small areas of suppuration at the center of which the ray fungus is found. Microscopically, there are present in the very vascular pyogenic granulation tissue many pale vacuolated cells which are not found in tuberculosis.[13] These cells may suggest the disease when the ray fungi cannot be found and more sections can then be taken and the responsible organism more closely searched for.

CLINICAL FINDINGS

The clinical findings in actinomycosis are those of any chronic suppurative process. Fever, loss of weight and appetite, pain, and weakness are part of the generalized picture.

There are usually many draining sinuses. Secondary infection may complicate the picture and conceal the presence of the ray fungus. Amyloid disease may result. The local symptoms will depend on the site of major involvement. Thus pulmonary actinomycosis may simulate tuberculosis or cancer in its clinical manifestations. Involvement of the gastrointestinal tract will simulate other granulomatous or neoplastic disease in this system. When the spine is affected back pain becomes an important feature. Neurological manifestations do occur, but are rare.

DIAGNOSIS

Recovery of the actinomyces in the discharges from the sinuses or their demonstration in histologic preparations are the only positive ways of making the diagnosis. Roentgen examination, however, may be helpful.

ROENTGENOGRAPHIC FINDINGS

The roentgen examination may aid in the diagnosis of actinomycosis before the vertebrae are involved especially where the lungs are the primary site. Kirklin and Hefke[6] have pointed out that the involvement of the ribs and sternum in association with parenchymal infiltration in the lungs, pleural thickening, and empyema more strongly indicates actinomycosis than any other process. The ribs and sternum may show destruction with or without the reaction of osteomyelitis or periostitis about them. If sinuses are present their connection with the destroyed rib need not be demonstrated. The laminagram will probably be of inestimable value in demonstrating lesions of the sternum.

When the vertebrae are involved the roentgen findings may be characteristic if not pathognomonic. Erosion of all portions of the vertebrae and adjacent ribs including the pedicles, spines, lamina and body is a feature. The body is usually affected in its cortical portion with periostitis as an early manifestation finally leading to erosion. The anterior surface of the body may have a saw-tooth appearance.[8] Reactive condensation takes place about destroyed areas and the vertebra involved may appear denser than its neighbors. The intervertebral disc is usually not involved. In no case is the uniform decalcification of one or more vertebrae found as is sometimes observed in tuberculosis.

Both tuberculosis and nonspecific osteomyelitis may on rare occasions simulate the appearance of actinomycosis of the vertebrae. However, involvement of the intervertebral disc with narrowing of the intervertebral space and finally collapse of the body of the vertebrae is the most common type of tuberculous involvement. Any deviation from this picture makes the roentgen diagnosis of tuberculosis difficult without supporting evidence from other sources. In nonspecific osteomyelitis biopsy and bacteriological examination must be resorted to, if the rest of the clinical picture is not conclusive. Even then difficulties may remain as actinomyces may not be present in a small biopsy specimen, and a prolonged and repeated search will be necessary before organisms are demonstrated.

CASE REPORTS

Case 1. J. S., Negro, male, aged twenty, was admitted to the Surgical Service on June 26, 1941, with a mass 4 cm. in diameter on the right side of the neck and another just to the left of the sternum. Both masses were definitely fluctuant, but not hot or tender. A draining sinus was present over the tibia. Another sinus on his head had healed. He had lost 25 pounds in weight in the five months prior to admission. As an inmate of a prison farm, he had contact with cattle and had chewed grasses and straws.

Physical examination on admission revealed a temperature of 38.8° C., a pulse of 130 per minute, and respirations of 22 per minute. The head was held to the right. The mouth and throat appeared normal. The teeth were in good condition. Aside from the masses described, the draining sinus on the left tibia, and signs of emaciation, the rest of the physical examination, including the neurological examination, was negative.

The laboratory findings showed the urine and blood to be normal. The Kline exclusion test was negative.

FIG. 1. Case 1. Anteroposterior view shows areas of destruction in the lumbar vertebrae.

Roentgen examinations revealed enlargement of the right hilum probably due to enlarged lymph nodes, but no definite evidence of pulmonary infiltration. There was an area of destruction in the right fourth rib posteriorly. The cervical spine appeared normal. There were areas of destruction in the left frontal bone. These findings were interpreted as osteomyelitis and mediastinal lymphadenitis of tuberculous origin. Roentgenograms of the vertebrae taken two months later revealed punched-out areas of destruction in the bodies, laminae, pedicles, and spinous processes of the vertebrae in the thoracic, lumbar, and sacral regions (Fig. 1 and 2). The intervertebral discs were intact. The vertebrae showed no evidence of collapse. The diagnosis was tuberculosis of the vertebrae.

The hospital course consisted of aspirations of the cervical mass from which thick yellowish-green pus, in which no acid-fast organisms could be found, was removed. He was given high voltage roentgen therapy to the cervical mass and to the areas of destruction in the skull. He was discharged August 27, 1941, the prognosis being poor.

The patient was readmitted to the Tuberculosis Service on December 3, 1941, because of an infiltration at the base of the right lung which appeared more definite than on the previous examinations. Aside from the diminution in the size of the cervical mass the patient had become worse. Physical examination revealed nothing new. The previous diagnosis was accepted and the patient was treated symptomatically with aspirations of the fluctuant masses. No tubercle bacilli could be isolated from the sputum or from the fluctuant masses. The patient grew gradually worse. The heart became enlarged and the possibility of a pericarditis was entertained. He died March 20, 1942.

At autopsy (performed by Drs. Lund and

FIG. 2. Case 1. Lateral view demonstrates that areas of destruction involve the laminae and spinous processes as well as the bodies.

Kapp) a large paravertebral phlegmon which contained thick, yellow, creamy pus was present in the thoracic and lumbar regions. The anterior aspects of the bodies of the vertebrae showed deep abscess pockets involving at least one-half of the depths of the bodies in most instances. The surrounding non-cavitated vertebrae were yellow, soft and crumbly. The phlegmon extended laterally over the lateral aspects of the vertebrae. Involvement of the ribs was not demonstrated.

Microscopically, much of the bone was replaced by areas of necrotic exudate. The cytology was not distinct, but the cells appeared to be lymphocytes and polymorphonuclear leukocytes. The areas were surrounded by a fibroblastic zone. In the necrotic areas were numerous ray fungi. The organisms were numerous and had peripheral radiating clubs and central net-like mycelia.

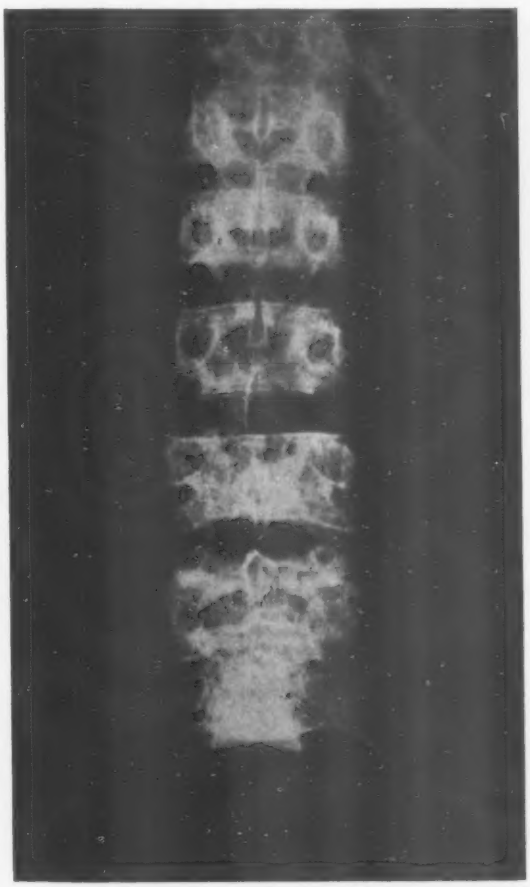

Fig. 3. Case II. Anteroposterior view shows widely distributed areas of destruction surrounded by areas of sclerosis.

Fig. 4. Case II. Lateral view reveals involvement of all components of the lumbar vertebrae. The areas of destruction are smaller but the sclerosis is more prominent than in Case I.

The final diagnosis was actinomycosis of lungs, vertebrae, and prevertebral tissues. The lesions in the skull, ribs and tibia were not examined.

Case II. H. M., white male, aged twenty-one, entered the Surgical Service of the University Hospitals of Cleveland on December 27, 1931, with complaints of pain in the back and draining sinuses in the right lower abdominal quadrant following an appendectomy at another hospital eight months before. Nothing unusual was noted in the appendix, but the diagnosis of actinomycosis had been made from the discharging sinuses.

Physical examination revealed a chronically ill patient. The teeth were in poor condition. There was spasm in both lower quadrants, especially on the right, and tenderness in the

region of the fifth lumbar vertebra. The neurological examination was negative.

The laboratory findings revealed traces of albumin in the urine and a white blood cell count of 13,000.

The diagnosis of actinomycosis was reaffirmed by another biopsy. Injections of the tracts with lipiodol and subsequent roentgenograms showed the tracts to end in the region of the cecum which was then removed surgically. Roentgenograms of the spine were negative. The sinus tracts were explored and through and through drainage instituted. The patient was discharged January 1, 1932, to be cared for at home.

The patient was readmitted November 13, 1932, with the complaint of convulsions, weakness and draining abdominal sinuses. During the interval at home he constantly grew worse. He had had two convulsive seizures and the legs had been held in a constant state of flexion for some time. There had been intermittent diarrhea.

Physical examination revealed a further increase in emaciation. The abdomen was diffusely tender and distended. There were five sinuses present in the right lower quadrant and two or three in the right lumbar region. The tendon reflexes were reduced. Sensation was normal.

The laboratory findings at this time were essentially unchanged.

Roentgen examination (Fig. 3 and 4) revealed multiple, round, radiolucent areas with surrounding sclerosis in the lumbar spine and sacrum and questionably in the ilium. The second, third, and fourth right transverse processes were markedly irregular as was the right twelfth rib. The intervertebral discs were not involved.

The patient had several convulsions while in the hospital and died eleven days after admission.

At autopsy (performed by Dr. Paul N. Harris) the abdominal organs were matted together by dense gray fibrous tissue in which abscesses could be seen. In the process of evisceration the periosteum was stripped off the vertebrae. The surface of the bone was rough and contained many small abscess cavities in which there was greenish-gray or yellow pus and destruction of the trabeculae of bone. Around the abscesses there was an increased amount of bone and the trabeculae were arranged circumferentially.

The anatomical diagnosis was actinomycosis of the left lung, liver, vertebrae, skin, subcutaneous and retroperitoneal tissues.

CASE III. J. D., white male, aged sixty-one, entered the hospital on the Medical Service on April 10, 1941, with the complaint of pain in the chest and chronic cough for two years. The pain was located in the lower portion of the left side of the chest and was most severe on coughing and deep breathing. The cough was productive only in the morning.

Physical examination revealed obvious loss of weight. The left pupil was smaller than the right. The teeth were in poor repair. Posteriorly in the left chest fine crackling râles were heard. An "e" to "a" change was heard posteriorly in the region of the fifth, sixth and seventh ribs just to the left of the spine. The liver was moderately enlarged. The spleen was not palpable. The reflexes were normal.

Laboratory examinations revealed no urinary findings of significance. The white blood cell count was 10,900.

Roentgen examination revealed a streaked infiltration extending outward from the left hilum which was interpreted as a bronchogenic carcinoma. Bronchograms revealed no abnormalities of the bronchi of the left lung.

The patient was bronchoscoped, but no mass or constriction in the left main-stem bronchus could be seen. An aspiration biopsy was noncontributory. He was treated by high voltage roentgen therapy and discharged on May 3, 1941.

The patient was readmitted on the Medical Service November 18, 1941. He continued to lose weight and his cough grew worse. His sputum had contained thick mucus, pus and occasionally blood. For the past several months he complained of a severe pain in the back.

Physical examination at this time revealed further emaciation. The lungs revealed a decrease in fremitus, râles at both bases and tubular breath sounds over the left chest. A dorsal kyphosis was present. The neurological examination was negative.

Laboratory findings were essentially unchanged.

Roentgen examination showed extension of the pulmonary infiltration on the left and an enlargement of the right hilum. The ninth thoracic vertebra (Fig. 5 and 6) was almost completely collapsed and there was questionable destruction of the adjacent ribs. The intervertebral discs were not involved. Bronchogenic carcinoma remained as the diagnosis.

The patient continued to grow worse, and developed cardiac irregularities, which were attributed to invasion of the heart by the neoplastic process. No further diagnostic procedures were done and the patient died on December 26, 1941.

At autopsy (performed by Dr. F. T. Kapp[6]) the lungs contained many soft-walled cavities filled with creamy material. Portions of the lungs were adherent to the chest wall, the mediastinum and the paravertebral tissues.

The vertebral column showed a kyphosis in the mid-thoracic region. The ninth thoracic vertebra was almost completely replaced by a cavity filled with a yellow, thick fluid. The spinal cord at the level of the ninth thoracic vertebra was soft and compressed by the collapsed bone around it. The dura in this region was surrounded by a thin, yellow layer of tissue.

Microscopic examination of lungs and lower thoracic prevertebral tissues showed the presence of a granulomatous and suppurative process containing the characteristic actinomycotic colonies. The body of the ninth thoracic vertebra was completely destroyed and the process caused a chronic inflammatory reaction in the outer layers of the dura in this region.

Fig. 5. Case III. Anteroposterior view shows collapse of the ninth thoracic vertebra.

Fig. 6. Case III. Lateral view shows sparing of the intervertebral discs despite the collapse of the ninth thoracic vertebra.

The final diagnosis was actinomycosis of the lungs and the ninth thoracic vertebra.

DISCUSSION

Three cases of actinomycotic involvement of the vertebrae have been presented. Two of the cases show the classical findings and one shows angular deformity which is only rarely described.[8] The first case probably represents involvement of the vertebrae secondary to primary mediastinal involvement. The involvement of the lungs is apparently secondary. The second case is

one of ileocecal actinomycosis with subsequent spread to the lumbar prevertebral tissues and vertebrae. The third case is one of primary pulmonary actinomycosis with spread to the mediastinum and vertebra. In the latter only one vertebra is involved in the roentgenogram.

In only 1 of the cases was there the contact with grasses and cattle. The other 2 patients did not have a history of rural occupations although the thoroughness with which this information was sought is not known.

In 2 of the cases the diagnosis was not made before death, either clinically or roentgenologically. In the case with the angular deformity of a vertebra, however, the roentgen findings were not characteristic, and even in review the diagnosis made seems to have been most proper. In the other case, the roentgen findings were sufficiently characteristic of actinomycosis to have suggested its presence. With this possibility in mind a biopsy or sputum examination might have revealed the actinomyces.

By the time the vertebrae are involved the disease is very extensive and the prognosis hopeless. All of the cases received roentgen therapy. The first case received small doses in a protracted course. The last case, however, received a full cancericidal dose.

SUMMARY

1. Actinomycosis is not necessarily restricted to persons with agricultural pursuits or other rural contacts.

2. Poor oral hygiene, a tonsillectomy, or a tooth extraction may more strongly indicate the presence of actinomycosis than a history of chewing grasses and straws.

3. When actinomycosis involves the vertebrae the roentgen findings usually are characteristic if not definitely diagnostic.

4. The roentgen signs of actinomycosis consist of areas of destruction in all portions of the vertebra, including the spinous processes and lamina, as well as the bodies, with varying amounts of surrounding sclerosis. Collapse of a vertebra despite extensive destruction is unusual, but even then the intervertebral discs are spared in the roentgenogram.

5. Three cases of actinomycosis of the vertebrae with roentgenograms illustrating the changes have been presented. Two of these cases present findings which are considered characteristic; the other shows angular deformity which is unusual.

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Vol. 51, No. 6

HEREDITARY FACTORS IN MULTIPLE CONGENITAL DEFORMITIES

REPORT OF TWO CASES

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CASE REPORTS

CASE I. Mrs. F.G., aged twenty-one, was admitted to the A. Barton Hepburn Hospital on March 1, 1941, and on the following day, gave birth spontaneously to an infant, N.R.G., having both feet clubbed and a fluid filled sac protruding from the midline in the lumbar region. The sac, incompletely covered by skin, was assumed to be a myelomeningocele. Roentgenogram of the region (Fig. 1) demonstrates defect of fusion of the sixth, eleventh, and twelfth dorsal and all of the lumbar vertebrae. The coexistence of craniolacuna was likewise verified by roentgenogram (Fig. 2). Death of the infant occurred on the ninth day. Postmortem examination confirmed the diagnosis of myelomeningocele. The cerebral ventricles were not enlarged.

FIG. 1. N.R.G. Showing spina bifida.

The maternal patient had never had an operation, serious illness or injury. She had first

FIG. 2. N.R.G. Showing lacunar skull.

menstruated at the age of fifteen, and had been habitually regular. She was married at the age of seventeen, and the pregnancy referred to above was her third. The first was terminated by miscarriage at three months and the second went to term, the product of conception being a monster with an encephalocele. During the current admission, Wassermann and Kahn tests were found to be negative, as were those of her husband. Blood cell counts were normal; there were transiently small amounts of albumin and sugar found in urine obtained by catheterization. Physical examination on admission revealed no significant abnormality.

During August, 1941, a fourth pregnancy terminated by miscarriage at four months. The

FIG. 3. Mrs. W.O., maternal aunt of N.R.G., showing spina bifida.

patient, again pregnant, came to the hospital on May 5, 1942, for roentgenographic examination, and a complete fetal skeleton was found in a position of favorable presentation. The maternal pelvis was gynecoid and the critical diameters appeared ample when measured by the Thoms grid method. On August 24, 1942, she was delivered of an infant having no physical or roentgenographic evidence of deformity.

In the family history, it was recorded that the patient's sister, Mrs. W.O., has had since birth a soft mass protruding from the lower part of her back, that she is otherwise in good health and has had two healthy children, both delivered by cesarean section. Through the courtesy of Dr. Joseph H. Wyatt of Newark, a roentgenogram was obtained (Fig. 3) demonstrating defective fusion of the second to fifth lumbar vertebrae. There was nothing else of significance in the family history. Neither parent of the sisters had ever been exposed to roentgen rays.

CASE II. Mrs. R.S., a primipara, aged nineteen, was admitted to the hospital on May 21, 1943, being then in labor. Roentgenographic examination disclosed an anencephalic fetus, which was subsequently delivered by dilating the cervix with a Voorhees bag. A postpartum roentgenogram of the fetus (Fig. 4) shows that, in addition to the deficiency of bones of the cranial vault, there is no fusion of the centers of ossification of the cervical spine. The maternal pelvis was found to be gynecoid and of ample dimensions. In the patient's past history there was only indefinite illness of childhood and pneumonia. Nothing of abnormal significance was found on physical examination or in the routine blood count, urinalysis or Wassermann reaction. In the family history, it is recorded that a second cousin (her mother's

FIG. 4. Anencephalic fetus with cervical spina bifida.

first cousin) had been delivered of an anencephalic fetus and that the patient's mother has six digits on each hand; her brother has six digits on one hand.

REVIEW OF LITERATURE

In presenting 2 instances of the occurrence of similar or related congenital deformities in members of the same family, the intent is to invite attention to recent medical literature offering a plausible explanation of the etiology of such deformities. Dorrance[5] attributes the defective development of the membranous bones of the calvarium in cases of lacunar skull to ischemia and increased intracranial pressure in the embryo. Bettinger[3] regards lacunar skull as a secondary effect of spina bifida, occurring when the pressure of cerebrospinal fluid over the cortex of the brain is reduced below the customary balance existing between it and the amniotic fluid, permitting the developing bones of the cranial vault to be compressed against the pulsating cerebral convolutions. The common association of craniolacuna and spina bifida is confirmed by Vogt and Wyatt,[8] who found craniolacuna to exist in 52 of 120 infants having meningocele, and 2 other cases of lacunar skull in which there was no meningocele. Shapiro and Tosti[7] concluded that dilatation of the cerebral ventricles is part of the picture of spina bifida with meningocele.

The unsupported fact of familial incidence suggests that spina bifida and the associated congenital lesions may be caused by a defect in the primary germ plasm. The comparative rarity of familial incidence would indicate that the genetic manifestation must be extremely recessive. In explanation of the mechanics involved in the formation of certain other congenital deformities Engel[6] offers a hypothesis based upon experimental observations of Bagg,[1] Bagg and Little,[2] Bonnevie,[4] and others.

Bagg and Little[2] exposed the gonads of healthy mice to moderate doses of roentgen rays and found that in the progeny of the irradiated mice there was a high ratio of animals having congenital defects of the head and extremities. These defects were carried on down through successive inbred generations though only the original progenitors received the experimental injury. The more common hereditary defects were of the eyes, varying from slight atrophy of the lids to opaque corneas and shrunken blind eyes with advanced atrophy of the optic tracts. There were somewhat less frequent defects of the extremities, including clubfoot, syndactyly and polydactyly. No such defect was found in more than two thousand control individuals of the same mouse strain. Not only was the fertility of the irradiated stock less than that of the control animals, but there were also many still-births. Blood vascular lesions were discovered about the heads of the embryos of the experimental mice.

In supplemental studies, Bagg[1] found that the embryonic lesions originally noted were at first clear fluid blebs beneath the surface, into which extravasation of blood often occurred. Blebs were found as early as the twelfth fetal day, the most frequent site being about the limb buds. Embryos were observed *in utero;* those individuals having blood blebs were marked by tail clipping, and the pregnant animal was allowed to proceed with gestation, with the result that the marked animals were found to be defective while those not so marked were usually normal. Occasional visceral lesions were encountered. Bagg concluded that irradiation injured the germ plasm, that the injury produced blebs containing blood which provided a mechanism for congenital malformation.

Bonnevie[4] carried on further experimental observations with animals bred from the Bagg and Little mouse strain. She found that the clear blebs contained cerebrospinal fluid that had escaped into the subcutis through the foramen anterius, an embryonic structure in the roof of the fourth ventricle. This structure is normally a safety valve guarding against increased cerebrospinal pressure in the embryos of vertebrates. It exists prior to formation of

the foramen of Magendie. In embryos developing blebs the amount of escaping fluid or the rate of flow is increased. It was observed that the fluid had a tendency to collect in the concavities of the embryonic surface and in the ends of the limb buds; that the blebs were apt to be damaging to further development of the embryo only if there was extravasation of blood into them. A preponderance of lesions of the left side was considered to be due to the intrauterine position of right-sided twist assumed by the mouse embryo. In outcrossing experiments, Bonnevie found that modifying genes changed the specificity of the deformity.

Engel[6] lists nine congenital syndromes in which the formation of spinal fluid blebs in the subcutis of the human embryo may constitute an etiological mechanism. He finds the present differentiation artificial, and notes that all of these syndromes have in common a combination of anomaly of the skull, deformity of the extremities and a tendency to familial occurrence. The specific conditions that he mentions are: oxycephaly, acrocephalosyndactyly, dysostosis craniofacialis, Lawrence-Moon-Biedl syndrome, Morquio's disease, pleonostosis familiaris, mongoloid idiocy, craniocarpotarsal dystrophy and Turner's syndrome. These are collectively called "bleb disorders," regarded as comparable with the experimental conditions produced in the Bagg and Little mouse strain. Engel suggests that still other conditions may eventually be included.

COMMENT

While two case reports can have no statistical value, they may be used to illustrate the applicability of a hypothesis. The second case rather strikingly illustrates in successive generations congenital defects similar to those experimentally produced in mice, though no single individual in the family exhibits malformation of both the head and the extremities. The first case herein cited conforms to all of Engel's stipulations for congenital disorders of bleb origin, in that there are deformities of the skull and feet and evidence of hereditary transmission. There are also fusion defects of the spine and a congenital disorder of the central nervous system. The relatively common spina bifida is not uncommonly associated with clubbing of the feet, and it has been shown that, when complicated by meningocele, there is apt to be craniolacuna. Such diverse manifestations can hardly be attributed to intrauterine physical conditions, nor can many of them be correlated as cause and effect. While craniolacuna has been regarded as a secondary effect of meningocele, its lace-like pattern is in fact not an image of the gyri of the cerebral convexity, and it is found at an earlier age than the appearance of the usual convolutional markings.[8] All of the manifestations of clubfeet, polydactyly, spina bifida with meningocele and defective cranial development may be explained by a mechanism of cerebrospinal fluid blebs extruded into the subcutis of the early embryo. The experimental and clinical observations appear to be analogous, and it is suggested that it is entirely plausible to include these defects occurring either singly or in combination along with the conditions listed by Engel as being of bleb origin.

The animal experiments causing inheritable deformity through injury to the gonads by roentgen rays have given rise to the supposition among students of genetics that the clinical use of roentgen rays may be responsible for congenital deformities occurring in human progeny. Such disorders cannot be entirely charged to roentgen-ray injury nor is it believed to be a principal etiologic factor since the deformities were commonly seen prior to the era of clinical radiology. The primary cause of clinical as well as experimentally produced hereditary deformities can be radiant energy or another agent capable of producing sublethal changes in the primary germ cell.

The author desires to thank Drs. F. E. Clark and R. L. Stacy for assistance in obtaining family history data.

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OSTEOCHONDRITIS OF THE CAPITELLUM (PANNER'S DISEASE)

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THE more commonly encountered sites and appearance of that interesting group of osteochondropathies, termed variously osteochondritis, osteochondrosis, aseptic necrosis, epiphysitis, and chondroepiphysitis, are well known to roentgenologists. Some of the rarer locations are understandably less familiar, and it is for this reason that this report is being made.

In 1927 Panner[1] first described the appearance of osteochondritis occurring in the capitellum, recognizing it as an entity belonging to the group that includes the more familiar sites described by Perthes, Köhler, Freiberg, Kienböck, Scheuermann, and others. In his published report[2] in 1929 he

FIG. 1

records 3 cases, 2 in boys aged ten and 1 in a boy aged seven. In a discussion of Panner's presentation, Krebs[3] reported a case of his own in a boy aged eight, and suggested the term "Panner's disease" for this condition. In 1929 Smith[4] reported a case in a four year old boy. Two cases described by Elward[5] in 1939 were both in eight year old boys. In a group of 91 endocrinopathic patients with osteochondritis in 162 areas, Schaefer, *et al.*[6] tabulate 3 instances of involvement of the capitellum. A careful search of the literature shows that these 10 cases are the only recorded instances of this condition. Doubtless it is not so rare as this would lead one to suspect, since its presence is probably overlooked in numerous instances. The following case was recently seen by the author:

D. S., male, aged eight, was perfectly well until three weeks prior to the examination. At that time, in the act of throwing a dart at the dart-board, he found he could not completely flex the right elbow. There was no previous history of any trauma. Since then he has developed a limitation of complete extension and complete flexion of the elbow (due to moderate

FIG. 2

discomfort) although the intermediate range of motion is free and painless, and pronation and supination are not interfered with. A slight swelling about the joint, particularly over the lateral aspect, has developed, but at no time has there been any redness. Figures 1 and 2 are views of the involved elbow, while Figures 3 and 4 show the uninvolved elbow for comparison.

From the data available in the recorded cases, osteochondritis of the capitellum may be summarized as follows: Clinically, the patient is a male (all of the cases in which sex was recorded were males) seven to ten years of age. There may be a history of a relatively mild preceding trauma or there may be merely a gradual onset of discomfort in the elbow. In the latter case the trauma may have been so insignificant that it is not recalled. The etiology of osteochondritis in general is still a debatable matter and is outside the scope of this paper. In any event, the discomfort is only moderate and accompanies chiefly attempts at extreme flexion or extension, which are limited. There is a slight swelling of the joint, but no redness.

The changes that are seen roentgenographically consist of an irregularity of contour of the capitellar epiphysis. The cortex is thin and there is a subcortical zone of rarefaction, as can be seen in Figures 1 and 2. There may be interruptions in the cortical continuity. Within the epiphysis

Fig. 4

Fig. 3

are seen irregular zones of bony condensation. In marked cases the epiphysis undergoes fragmentation. These changes are the ones generally seen in osteochondritis in any site. The epiphysis, as a result of these alterations, may appear smaller than normal and the joint space between the epiphysis and the head of the radius appears greater than normal. Some soft tissue swelling may be seen about the joint, especially on the lateral aspect. This condition once again demonstrates the advisability of making bilateral examinations of the joints in children, since the changes are readily apparent when compared to the normal, but if the condition is an early one, it can easily be overlooked by someone not completely familiar with the normal appearance of the epiphysis.

With rest and relative immobilization af-

forded by a sling, the clinical symptoms and signs disappear over a period of a few weeks. The roentgen manifestations are not commensurately improved, however, and may persist for one to three years before complete restitution is seen. This benign course resulting in practically complete restoration of structure and function follows the familiar pattern in osteochondritis where weight bearing is not a factor. Theoretically, however, such an elbow may possibly be more susceptible in later years to the development of secondary hypertrophic or traumatic arthritis. No case has been followed long enough to determine this.

SUMMARY

Attention is called to an apparently rare site of osteochondritis, the capitellum, and another case is added to the very few reported in the literature.

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ROENTGENOLOGICAL FINDINGS IN BILATERAL SYMMETRICAL THINNESS OF THE PARIETAL BONES (SENILE ATROPHY)

REPORT OF A CASE WITH A REVIEW OF THE LITERATURE

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BILATERAL symmetrical thinness of parietal bones, also known as senile atrophy of the parietal bones, biparietal thinning, symmetrical thinness of the parietal bones, and so forth, is well known in the anatomical, anthropological and pathological literature but has received little attention in that devoted to roentgenology.

HISTORICAL

In all probability the condition was known to the ancients, since many examples taken from Egyptian tombs have been described.[14,48,56,57] Chiari[9] ascribes the first modern description to Köhler[29] in 1786. Descriptions began to make their appearance in the European literature with Voigtel[62] in 1804 and thereafter increasing numbers of reports appeared. Ribes[42] in France and Humphry[23] in England were among the early writers. In the Western Hemisphere, Shepherd,[54] first as Demonstrator and later as Professor of Anatomy[55] at McGill University recorded the first cases found in North America. Willard[66] described the first cases found in the United States in 1905. It was not until 1926 that Moore[34] in this country and Casati[7] in Europe published papers with illustrations of roentgenological findings.

SITE OF INVOLVEMENT

The characteristic location of cranial thinning is in the parietal bones between the sagittal suture and the parietal prominence. Earlier authors, notably Humphry,[23] claimed it could occur only in that region. However, subsequent experience has proved this contention erroneous; in Bloch's[4] case, both temporal bones were involved and Chiari's[9] had implication of the parietal, temporal, frontal and occipital bones on both sides. Greig[19] illustrates one example of thinning of the planum occipitale and Casati[7] shows a solitary depression which crosses the sagittal suture somewhat anterior to the bregma. The latter is the only example we have been able to find in which the sagittal suture was actually crossed; in no case of bilateral thinning has this occurred.

Characteristically there is a crest-like ridge of intervening bone, centered on the sagittal suture which separates the parietal depressions; frequently there is a shallow groove-like depression of the sagittal crest —this feature is illustrated in the postero-anterior roentgenogram of our case.

TYPES

Greig[19] described two chief forms: (1) a roughly triangular or quadrilateral flattened depressed area, and (2) a trough-like depression running longitudinally along both sides of the sagittal suture; in the latter type, extension onto the frontal and occipital bone is likely to be found.

PATHOLOGICAL CONSIDERATIONS

There is no sharp line of demarcation between the depression and adjacent normal external tables; the margins shelve gradually into the thinned area. The walls of the depression are usually dark due to blood-containing medullary spaces,[67] and may be porous, though usually there is sclerosis. The floor of the depression is usually white, in sharp contrast to the darker color of the walls, and frequently it is of parchment-like thinness, extremely fragile and translucent. An excellent example of this feature is shown by Pancoast, Pendergrass and

Schaeffer[38] in a specimen from the Warren Museum of the Harvard Medical School.

Humphry,[23] Durward,[13] Greig,[19] and Hill[21] have drawn attention to the fact that the parietal foramina are spared; upon approaching them, the thinning process skirts around and leaves a margin of bone about a centimeter in width.

Penetration of the thin floor may take place with consequent exposure of the dura. Virchow[61] was probably the first to point out that the process commences in the superficial layers of the external table and gradually progresses through it, the diploe and, on occasion, the inner table. Previously Rokitansky[45] and Lobstein[30] thought that the diploe disappeared first, thus allowing the inner and outer tables to come together and coalesce. Sauvage[48] thought that the process started with the disappearance of the meningeal lamina of the inner table; thereafter it caused absorption of the layers of the external table next to the diploe whence it spread through the more superficial layers of the lamina externa and then to the diploe. There appears to be little support for his ingenious theory. Virchow's explanation gains support from the fact that the defects are always widest at their external lamina aspect.

Virchow[61] was the first to study ground sections microscopically, and his findings have been confirmed in more recent years by Ziegler[67] and Schmidt.[50] According to them the process starts by localized disappearance of the superficial layers of the external lamina. Immediately beneath the absorbed region the bone tissues become more stratified than usual and become sclerotic by apposition of lamellar bone spicules in the diploic spaces. Subsequently this stratified sclerotic zone disappears by absorption and that which was normal, lying immediately beneath, becomes sclerosed. In this manner the process extends progressively through all the cranial layers.

Schmidt[50] was impressed by the lack of osteoclasts and came to the conclusion that the process is one of diminished bone formation rather than of increased bone destruction.

ETIOLOGY

Various theories have been advanced in attempts to explain this curious thinning. Ferré[16] states that Oribase and Fallopius attributed the thinning to baldness, the deficiency of hair permitting emaciation of soft parts and of bone. By the majority of authors it is considered an atrophic change, but explanations as to the cause of the atrophy vary widely.

Virchow[61] is generally credited with being the first to suggest the theory of senile atrophy. As a matter of historical fact that distinction belongs to Lobstein,[30] and Virchow recognized his priority; Rokitansky[45] and Maier[31] both adopted this theory before Virchow. Many other authors[7,16,25,28,37,50,54] have felt it the logical explanation. In the main, evidence of skull senility has been felt to depend upon (*a*) marked degree of decalcification, (*b*) generalized diminution in width of the tables, (*c*) suture obliteration, (*d*) edentulous condition of the maxilla with atrophy and (*e*) osteoporosis of the skull tables. Ashley-Montagu,[1] in a full discussion of the question of skull aging, concluded that suture closure and state of dentition are the more utilizable guides for determining age. Topinard[59] found parietal thinness and non-closure of sutures in the skull of a man between sixty and seventy years of age; Greig[19] found thinness of both parietals in a skull which still showed traces of the metopic suture and had full dentition in a good state of preservation. In our case all sutures, including the metopic, are visible and the upper teeth are well preserved. It would thus appear that the "senile" theory cannot be accepted as the cause in all cases.

As a variant of the senile theory, Carriere[6] and Weber[63] considered it due to excessive physiological atrophy. The latter concluded that the involved area has no muscle attachments and is mechanically of little value "for maintaining the strength of the brain case."

Syphilis, as might be expected, has been held responsible by many authors. Paget[36] was among the first to form this opinion but he later became converted to the senile atrophy theory. Rokitansky,[45] Topinard,[59] Hutchinson[27] and Meyer[32] later adopted this theory, though Meyer was not enthusiastic. Hutchinson appears to have had some justification because his patient was known to have had a chancre. Modern writers,[41,43] reporting serological findings in their cases, have found no evidence of lues, and in our case the Wassermann and Kline reactions were negative.

Sauvage[48] based his views upon the study of 41 cases which formed the basis for his thesis and concluded that interference with vascular supply was the probable cause. In 1870 Carriere[6] expressed the opinion that the change is due to atheromatous vascular changes. Broca[5] lent some support to this theory when he stated that the posterior parietal region is the last to ossify and that vascular nutrition is least in this part of the skull. Shepherd,[55] in attempting to explain the vascular theory, held that the temporal arteries which supply the external table, being uncovered by muscle, are more apt to become involved by senile change and so interfere with nutrition. This type of change is less likely to affect the middle meningeals which supply the inner table. Durward[13] and Hill[21] were inclined to support this theory. In comment it should be observed that there have been no reports of careful dissection and microscopic studies of the vascular supply to the parietal bones in cases of thinning. Until such studies have been made, the theory is open to question.

Humphry[23] proposed the theory of congenital defect but later[25] acknowledged he had insufficient evidence to support it, and favored that of senility. Shepherd[55] had a case, unproved, in a woman who stated that her depressions had been present since she could remember and that her father had had similar depressions. He concluded that the condition could be both congenital and hereditary. Bloch,[4] and Roger and Schachter[44] have been among those who accepted this theory.

Chiari[10] stated that Després[12] originated the theory that "planing" action of the galea aponeurotica across the parietal eminences might cause a gradual wearing away of the external table. Humphry,[26] abandoning his two previously held theories, evidently believed this viewpoint original with himself. Chiari[10] concluded that this was the most probable explanation. Greig,[19] on the contrary, considered it beyond the power of the aponeurosis to produce such a change and G. E. Smith[56] was of the opinion that the parietal bones would be more likely to modify the tendon. The fact that the defects occur in parts other than the parietal bones is further evidence against this theory.

Meyer,[33] Rossbach,[46] and Hollander[22] were the chief exponents of the theory that mental disorders, notably melancholia, can cause the deficiencies. While unbiased writers have reported mental instability[43] and headaches,[34,41] and though our patient is subject to attacks of vertigo, the number of patients showing reliably reported mental signs is too few to consider this the sole cause. Moreover it is not unlikely that the symptoms in many cases are a manifestation of the individual's period of life.

Smith[56] found 70 cases among Egyptian mummies of the wealthy class who lived between the Fourth and Nineteenth Dynasties. He noted that people of that class and in that period were in the habit of wearing heavy, full-bottom wigs and concluded that continuous pressure from the weight of such an object was the cause. While this may have been the cause in the particular class of people to whom he had reference, it cannot be considered so among the cases from Europe and this country reported since the middle of the nineteenth century. A further objection lies in the fact that the thinning does not always occur at the parietal eminences.

Greig[19] has been followed by Cave[8] and

Pancoast, Pendergrass and Schaeffer[38] in support of the so-called "dysplasia" theory. According to this view "in the centre of the affected parietal area the diploe has never developed, and this part of the parietal has remained in the same condition as the lower parts of the inferior occipital fossae where the bone is thin, semi-transparent and destitute of diploe" (Greig). Later in life, as the surrounding diploe develop, the area of abiotrophy[19] remains as a depression. Greig's arguments are convincing and his theory could explain the known cases.

INCIDENCE

The abnormality is not confined to the human race. While Pick's[40] cases among dogs were of the generalized type affecting all of the skull bones, Humphry's[24] example in an adult female orang-outang was typical of the groove or trough defect found in man.

Racially, a large number of cases have been reported among ancient Egyptians;[14,48,56] this preponderance is probably more apparent than real since large quantities of concentrated material have been made available through archaeological investigations. Many cases have been reported among Europeans of England, France, Italy and Germany.[25,19,27,48,15,16,7,41,34,10,61] In the United States, Moore,[34] Meyer,[32] and Willard,[66] and in Canada, Shepherd[54,55] have reported cases. Durward[13] had 3 cases among the aborigines of New Zealand and 2 in Chinese. His 2 cases, with ours and that reported by Davis and Thurnam[11] comprise the 4 cases found among this race. Hill[21] had a case in a Singhalese. In all probability the condition is more common than is generally realized and doubtless more cases will be reported from time to time. There appear to be no racial limits.

Regarding incidence according to sex, it is difficult to draw accurate conclusions. More than two hundred cases are included in the literature reviewed herein, and in only 69 was the sex recorded; of these, 45 occurred in females. It is quite possible that accurate records might either modify or reverse the proportions.

According to age, Shepherd's[55] case in a forty year old male appears to be the youngest of those whose age was definitely known. Smith[56] found examples in skulls which he considered to be not under twenty-five or possibly thirty years of age. The upper limit seems to be restricted only by the natural span of life; Sauvage[48] had one example in an individual who had attained eighty-eight years.

FREQUENCY

Durward[13] found 5 cases in one thousand dissected skulls. Sauvage, in an investigation of the museums of Paris, found 28 examples in two thousand skulls. Smith and Jones[57] found 7 cases in ten thousand skulls. During the past five years we have seen skull roentgenograms of more than five hundred different patients of various age groups and this is the first example to come to our attention. While statistically our material cannot be considered sufficient to be significant, the conclusion appears to be warranted that the condition is not common among the average population of this country.

IMPORTANCE

While, in view of its apparent rarity, the abnormality under consideration may be considered as more of a curiosity than of clinical importance, two writers[26,66] have reported cases of death following fracture through such thinned areas. A comparatively trivial head injury might conceivably result in extensive brain laceration from penetration of fine splinters of bone. A head injury which would cause no more than a bruise in an ordinary individual might result in death to one whose skull had these thin spots. The legal complications could be extremely involved and interesting.

ROENTGENOLOGICAL CONSIDERATIONS

There is a singular dearth of references and illustrations in the roentgenological literature. Schüller[51,52] and Grashey[18] state

that interesting roentgen appearances may be found but do not include illustrations. Hempel[20] does not mention it at all. Roger and Schachter[44] associate the thinning with parietal lacunas in a descriptive paper but do not show illustrative roentgenograms. Pancoast, Pendergrass and Schaeffer[38] show photographs of specimens but do not include roentgenograms.

Moore[34] and Casati[7] illustrated their reports with reproductions of roentgenograms but showed only frontal views in which a thin bridge of bone covered over the defects. Judged upon the basis of their illustrations the cases could, with justice, be considered examples of enlarged parietal foramina but for the fact that the individual patients were adults, and, in such, enlarged parietal foramina are usually sharply defined, circular radiolucent areas.[38] (Fig. 5)

Raviola[41] and Rizzo[43] illustrate their case reports with reproductions of roentgenograms identical in appearance with ours in both posteroanterior and lateral projections.

Fig. 1. Tangential view of specimen No. 12640 in Warren Museum, Harvard Medical School. Compare with Figures 3 and 4. (Figures 1 and 2 are reproductions from roentgenograms furnished the author by Drs. M. C. Sosman and M. S. Donovan.)

Fig. 2. Superior-inferior view of specimen No. 12640 in Warren Museum, Harvard Medical School. (The photographs of this specimen are reproduced in Pancoast, Pendergrass and Schaeffer,[38] page 11, Figures 13 and 14.)

Since the illustrations of Moore, Casati, Raviola and Rizzo were from living persons upon whom no dissection studies had been done, we felt it advisable to prove beyond reasonable doubt that our case is indeed bilateral symmetrical thinness of the parietal bones. Since our patient is now in excellent health and prospects of autopsy proof consequently remote, it became apparent that the diagnosis would require support from analogy to known anatomical material upon which roentgenographic studies had been carried out. Through the great kindness and cooperation of Dr. Merrill C. Sosman and his associate, Dr. M. S. Donovan, I was furnished with tangential and superior inferior roentgenograms of the specimen (No. 12640) in the Warren Museum of Harvard Medical School; the photographic illustrations of this skull cap are published in Figures 13

and 14, page 11, of "The Head and Neck in Roentgen Diagnosis."[38] Photographs of these roentgenograms are reproduced herewith as Figures 1 and 2. By comparison with our Figures 3 and 4, the similarity will become apparent.

DIFFERENTIAL DIAGNOSIS

In a given roentgenographic study it will become necessary to differentiate symmetrical bilateral thinness of the parietal bones from important pathological conditions. Neoplastic disease may be excluded from the finding of (1) a symmetrical bilateral localized involvement with (2) smooth, regular margins, (3) absence of a surrounding zone of new bone formation, (4) lack of evidence of malignant lesions elsewhere in the body and (5) lack of pain or tenderness. Pressure erosion of an intracranial tumor would cause the defect to be wider at the inner than at the outer table of the skull, and it would be unusual to find the defects symmetrical as is the case in bilateral thinning; furthermore, in disease so advanced one would expect pronounced intracranial roentgenological manifestations with symptoms referable to the brain. In bilateral symmetrical thinness there is no increase in skull size, increased thickness of skull tables or "woolly" appearance characteristic of osteitis deformans. In addition, the skeleton elsewhere will be negative for evidence of this disease. Syphilitic involvement may be excluded by absence of positive serological findings, absence of porous or "moth-eaten" bone appearance, sequestration and periostitis. It is to be differentiated from osteomyelitis by lack of history of preceding inflammatory disease of the scalp, sinuses, mastoid or osseous system elsewhere and from lack of history of skull trauma. There is no periostitis, sequestration or surrounding bone reaction in bilateral symmetrical thinness, and fever, pain and tenderness are absent. From congenital defect such as osteogenesis imperfecta it may be differentiated by the appearance of bone which presents the impression of having become modified locally after having been normally formed, by no increase in width of the sutures, absence of enlargement of the head and lack of abnormal skeletal changes. Examination of the scalp with determination of absence of operative scars will suffice to exclude the possibility of trephine openings.

Enlarged parietal foramina differ in that they are openings through both tables of the skull and more radiolucent than thinned areas. The parietal foramina are usually situated nearer the midline than are areas of symmetrical thinning and on palpation, pulsations may be felt. Enlarged parietal foramina show a hereditary tendency,[38] and this may be the most important differential feature when one has to deal with diagnosis of a case of bilateral thinness in which both tables have become perforated.

CASE REPORT

C. L. W., a male Chinese, aged sixty-nine, was admitted to St. Vincent's Hospital on November 7, 1941, as a patient of Dr. George Schenck, after having been struck by an automobile. He sustained fractures of the sixth and eighth right ribs, an injury to his right hand, lacerations of the scalp, and shock. The presence of rib fractures was confirmed by roentgenological examination; there was no fracture of the hand.

He was born in California but as a young man had lived for many years in China where, according to custom, he had worn a pig-tail. However, for the past thirty years he had lived in Norfolk. He had been in excellent health all his life and had never before been a patient in a hospital. About three months prior to admission he had had his lower teeth extracted because of caries. For about three years he had had attacks of vertigo which came on while walking. Inquiry into bodily functions failed to elicit any indication of abnormality; specifically he denied any dysuria, digestive, pulmonary, cardiovascular or neurological symptoms.

He has three sons, one* of whom lives in

* This son is forty-five years of age and was born in China. He has always been in good health and owns his own laundry business. To palpation he presented posterior parietal "soft spots" similar in all respects to those of his father except that they were smaller. For superstitious reasons he has persistently refused to permit roentgenological examination of his skull.

Portsmouth, Virginia, the others live in California.

Blood pressure on physical examination was 114/72. Temperature, 96° F.; pulse 74, regular in rate, force and rhythm; respirations 26.

Apart from tenderness associated with the rib fractures and consequent limitation of respiration on the right side, the thorax, lungs and heart showed no abnormality. The abdomen was soft, without localized tenderness or masses. The prostate was small and fibrosed. The penis was normal as were also the testicles. The eye grounds were normal and there was no abnormality of neuromuscular reflexes.

The interesting physical findings were in connection with the skull. On either side of the midline, in the posterior parietal region there were palpable two "soft spots" each measuring approximately 4.5 by 6.5 cm. in greatest diameter, having their long axis parallel to the sagittal line. To palpation they gave the impression of being depressions whose margins shelved rather abruptly downward; there was no surrounding elevation of bone. In their central portions one palpated a pad of soft tissue but from the sense of resistance one received the impression that they had a bony floor. There was no localized soft tissue swelling over these areas and the patient made no complaint of tenderness on palpation. No pulsations or fluctuation could be detected and, when steady pressure was applied over them, there was no alteration in blood pressure. These "soft spots" were situated between the midline and parietal prominence on either side, were symmetrical in location and were separated from each other by a bony ridge approximately 5 cm. in width which centered on the sagittal suture. There was no evidence of scar from a surgical operation anywhere in the scalp. The hair showed a tendency to thinness over the frontal and temporal regions but was moderately thick over the parietals and occipital.

FIG. 4. Basal (parieto-occipital) roentgenogram of the case reported here. Note similarity to Figures 1 and 2.

FIG. 3. Posteroanterior (nose-forehead) roentgenogram of the case reported here. See text for detailed description.

In response to direct questioning the patient stated that he had known about these "soft spots" for two or three years and he associated them with his attacks of vertigo. It was not possible to prove any connection and no cause was found for his stated unsteadiness.

The skull and scalp were otherwise negative for any abnormal findings on clinical examination. The maxillary teeth were present and, apart from crowding of the central incisors,

FIG. 5. Lateral roentgenograms of the case reported here.

were apparently not abnormal; the lower jaw was edentulous, and showed no evidence of inflammation or other abnormality.

Because of persistent vomiting, frontal scalp swelling associated with lacerations and vague head pain—all attributed to the accident—the patient was referred to me for roentgenological examination of the skull. The following is a description of the findings which are illustrated in Figures 3, 4 and 5.

In posteroanterior (nose-forehead) and basal (parieto-occipital) projections there were bilateral excavations of the external table and diploe, symmetrically placed on either side of the midline and separated by apparently normal bone approximately 6 cm. in width. These depressions were about midway between the sagittal suture and parietal eminences. The depression on the left side was slightly broader than that on the right—5.0 cm. and 4.5 cm. respectively. The depth, from pericranial line to floor, was 2.0 and 1.8 cm. for the left and right depressions, respectively. The floor of each depression was represented by crossing convex-concave linear bone shadows, considered to represent the superimposed meningeal surfaces of the inner table and the floor of the depression; no evidence of perforation could be detected. There was a shallow, midline grooving of the sagittal suture and adjacent bone on either side, measuring approximately 1 cm. in width by about 3 mm. in depth.

In lateral projection (Fig. 5) the defects were visible as ovoid zones of increased radiolucence in the posterior parietal region on either side; each was about 6.5 cm. in sagittal length. The margins were not sharply defined but appeared to shade gradually into the density of surrounding skull tables. The defects were smooth in outline and presented no localized irregularities; there was no evidence of periosteal reaction or of sequestration.

Just anterior and medial to these defects there were small, irregular areas of increased radiolucence which were judged to represent depressions for pacchionian bodies; there was no evidence of connection between these and the larger defects described above.

In the frontal region the calvarial thickness was approximately 0.8 cm.; over the depressed areas (in lateral projection) it was approximately 1.0 cm., and from that point to the inion there was a gradual increase in thickness. At no point was there any suggestion of abnormal skull thickness. There was no irregularity of calcification, and nothing indicative of osteitis deformans, neoplastic or inflammatory disease could be seen. A few localized areas of rarefaction, judged to be indicative of slight osteoporosis, were seen.

There was no evidence suggestive of fracture, either recent or old.

By strong transmitted light the scalp outline could be seen in the original roentgenograms, and there was no evidence of local increase in thickness; over the parietal regions lateral to the depressions it was approximately 0.6 cm. and over the sagittal crest it was approximately 0.9 cm. thick. Soft tissue filled in the depressions but there was no bulging above the external scalp line.

There was no evidence of increased intracranial tension. All of the sutures, including the metopic, were visible and not widened. The diploic vessels were not unduly prominent nor were their grooves unusually deep. The sella turcica measured 0.8 cm. in depth and 1.2 cm. in length and showed no evidence of erosion. The pineal body was not sufficiently calcified to be visible.

The paranasal sinuses showed no indication of abnormality other than moderate thickening of the maxillary antral mucosa. The maxillary

teeth were present; the mandible was edentulous.

Roentgen examination of the entire skeleton failed to show any evidence of inflammatory, neoplastic or other pathological involvement. Particular attention was paid to the spine, pelvis, femurs and tibiae but no evidence of osteitis deformans could be detected.

There were bilateral renal and right ureteral calculi; the solitary left renal calculus was in the lower superior minor calyx and measured 0.4 by 0.6 cm. There were two right renal calculi, both in inferior minor calices; the smaller (lower) one was 0.3 cm. in diameter and the larger, about 1 cm. above it was 0.4 by 0.9 cm. in size. The left renal drainage system was sharply defined and was judged to be normal. There was slight dilatation of the superior calices and renal pelvis on the right side. The right ureteral calculus measured approximately 0.9 by 1.5 cm.; it was ovoid in outline and had caused dilatation of the ureter down to a point opposite the middle of the sacral wing; it was movable within the ureter. Dr. Devine noted no obstruction on catheterization of this ureter, however. The renal shadows were normal in size and the psoas shadows were sharply defined.

The patient persistently refused to admit tenderness of either renal or ureteral region and denied any symptoms referable to the urinary tract.

Culture of urine from the left kidney was sterile after forty-eight hours; the right specimen showed *Staphylococcus haemolyticus* after forty-eight hours.

Roentgen examination of the lungs and heart revealed no abnormality; there was no suggestion of pulmonary tuberculosis or neoplasm.

The gastrointestinal tract was investigated roentgenologically by ingested barium sulfate suspension and by barium enema. There was no evidence of malignancy or other abnormality.

Clinical Course. The patient's recovery was somewhat slow, chiefly owing to the fact that he could not make himself understood and could not properly understand what was said to him without the aid of an interpreter. On November 22 he had a sharp rise of temperature to 105° F., pulse to 120 and respirations to 36. It was thought that he had some respira-

Laboratory Findings.

Blood	Nov. 8	Nov. 12	Nov. 23
Hemoglobin	90%	90%	91%
Erythrocytes	5,500,000	4,600,000	4,880,000
Leukocytes	10,100	13,900	37,850
Polymorphonuclears	71%	71%	71%
Large lymphocytes	22	22	7
Stab	3	4	14
Transitionals	3	2	Myelocytes 5
			Metamyelocytes 3
			Marked toxic granulations

Sedimentation rate	1st hour	40
	2nd hour	60
Blood calcium	Nov. 12	10.0 mg.
	Nov. 16	11.0 mg.
Wassermann and Kline	Negative	

Urinalysis—all specimens voided.

	Nov. 8	Nov. 12	Nov. 18	Nov. 24
Color	Yellow	Amber	Amber	Cloudy, red color
Reaction	Neutral	Acid	Acid	Alkaline
Specific gravity	1.008	1.015	1.015	1.017
Albumin	Negative	Negative	Trace	Trace
Sugar	Negative	Negative	Negative	Negative
Mic. pus			Few	
Mic. blood			Occasional	Few
Epith.	Occasional			
Crystals				Triple phosphate and amorphous phosphate

tory distress and a diagnosis of pneumonia was considered but could not be proved by clinical or roentgen examinations. The cause apparently was dietary since, when fluids were forced and he was placed on a high caloric diet, immediate improvement resulted and his temperature, pulse and respirations returned promptly to normal. Careful studies failed to reveal any etiological factors responsible for the febrile state of November 22 or the marked leukocytosis recorded on November 23. On the latter date his temperature, pulse and respirations returned to normal and his recovery thereafter was uneventful. He was discharged from the Hospital on December 1, 1941, asymptomatic and afebrile.

He was seen again on February 19, 1942, at which time he stated he felt perfectly well and had no complaints. The findings relative to the softened areas of the parietal regions had not changed in the interval.

COMMENT

The above case is interesting because of the large symmetrical thinned areas in both parietal bones. Worthy of note are the facts that, with the exception of urinary lithiasis and right pyelonephritis, no possible cause was found; it is doubted that there is any etiological association between the urinary tract and cranial abnormalities. The possibility of similar cranial involvement in a forty-five year old son requires further investigation. This will be undertaken at the earliest possible opportunity. Serological studies were negative for evidence of luetic involvement and the blood calcium level was normal on two occasions.

If one accepts the state of dentition and non-closure of sutures as being evidence of lack of senility in the skull, the above case may offer additional evidence against the theory of senile atrophy. It is also worthy of note that the larger defect in our case was on the left side. This is contrary to Sauvage's[48] statement that the right side is usually more extensively involved. The possibility of association between the wearing of pig-tail in youth and the development of thinned areas is extremely unlikely.

SUMMARY

1. A case of bilateral symmetrical thinness of the parietal bones has been presented.

2. Evidence in support of this diagnosis has been submitted; briefly this evidence consists of:

(*a*) Analogy between roentgenological findings in a known specimen in the Warren Museum of Harvard Medical School and the roentgenological findings in our case.

(*b*) Similarity between our case and others reported in the roentgenological literature.

(*c*) Exclusion, in our case, of pathological processes likely to cause such defects, elsewhere in the body.

3. The above is, to the best of our knowledge, the second case with roentgenological findings to be published in this country, and the fifth with such findings in the world literature.

4. The anatomical, pathological, anthropological and roentgenological literature* has been reviewed and briefly recorded from the standpoints of (*a*) historical, (*b*) pathological, (*c*) etiological, (*d*) incidence, (*e*) frequency, (*f*) importance, (*g*) roentgenological, and (*h*) differential diagnostic considerations.

The author desires to express his thanks and deep appreciation to Dr. Merrill C. Sosman and Dr. M. S. Donovan, Peter Bent Brigham Hospital, Boston, for supplying roentgenograms from which Figures 1 and 2 were made; to Drs. John D. Camp and Leo A. Nash for kindly comment and a pre-publication "work" copy of their paper; Dr. George Schenck, Norfolk, Virginia, for permission to use this case; Dr. C. J. Devine, Norfolk, Virginia, for cystoscopic examination and ureteral catheterization preliminary to retrograde pyelography; Rev. Sidney Quong, Norfolk, Virginia for aid as interpreter.

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THE ENCEPHALOGRAPHIC APPEARANCE IN TWO CASES OF PONTINE GLIOMA IN CHILDREN

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IN GLIOMA of the pons the early signs are cranial nerve palsies, and increased intracranial pressure is a late development. For this reason, the neurologist, at times, has difficulty in distinguishing between cases of glioma of the pons and encephalomyelitis. At the Cleveland Clinic encephalography has been performed in 2 cases of glioma of the pons, and the appearance was found to be characteristic.

The 2 cases in which we are reporting the pneumo-encephalographic appearance were children three and eight years of age. In both of the cases the diagnosis was either encephalomyelitis or a pontine neoplasm. There was no evidence of increased intracranial pressure in either case, and accordingly air encephalograms were made.

The three year old girl (Case 1) died in this institution a short time later, and a postmortem examination confirmed the encephalographic diagnosis. There was a marked nodular tumefaction of the pons, more pronounced on the right side. Sections through the pons and cerebellum showed marked swelling of the pons, causing a posterior dislocation of the floor of the fourth ventricle. Microscopic sections showed the tumor to be a spongioblastoma polare. This specimen is shown in Figure 4*A*.

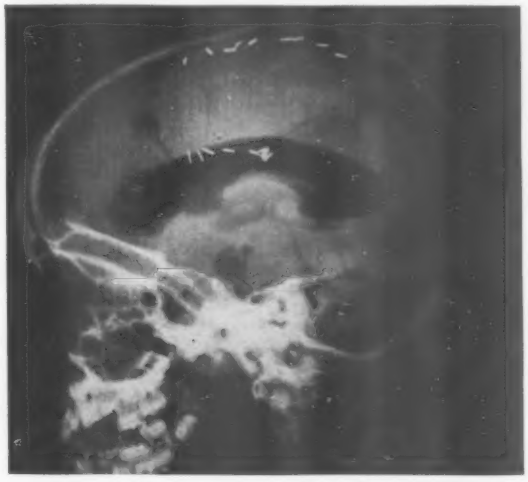

FIG. 1. This encephalogram was selected because it depicts well the normal contour of the pons. It is the case of a child who had been operated upon for a subdural hematoma. The pons is well outlined, bounded posteriorly by the fourth ventricle and anteriorly by the pontine cisterns. The thickness of the pons is 2.3 cm. The floor of the fourth ventricle is flat or perhaps slightly concave, and the normal curve of the aqueduct is well shown.

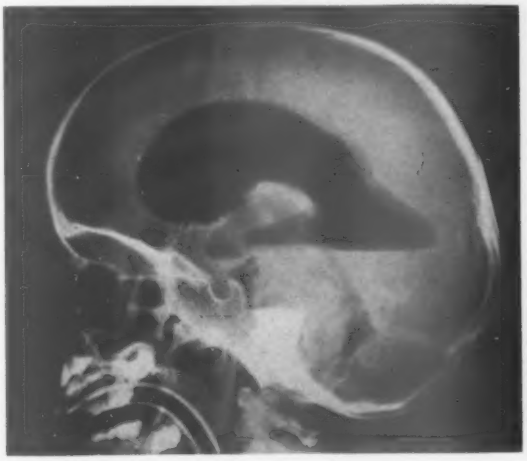

FIG. 2. Case 1. There is a marked dilatation of the entire ventricular system with some flattening of the pontine cisterns and no air in the subarachnoid space above the interpeduncular cistern. The thickness of the pons is 3.6 cm. The floor of the fourth ventricle is convex; the curve of the aqueduct is less acute than normal, and there is an upward bulging of the posterior portion of the floor of the third ventricle.

The parents of the second child (Case 11) refused to accept a fatal prognosis and took the child to another hospital where he was operated upon and died a few months later. The pathological report of a biopsy was pontine glioma. No postmortem examination was made in this case.

The pneumo-encephalographic appear-

FIG. 3. Case II. There is a moderate dilatation of the entire ventricular system with no air in the subarachnoid space. The thickness of the pons is 4.2 cm. The pontine cisterns are obliterated; the floor of the fourth ventricle is convex; the curve of the aqueduct is less acute than normal; and the tumor has caused a slight bulging of the posterior portion of the floor of the third ventricle.

ance of pontine tumors has been described by Lysholm,[1,2] but we feel the films in these 2 cases are so characteristic that they should be reported.

In the normal encephalogram the thickness of the pons, including the basilar and tegmental portion from the roof of the cisterna pontis to the floor of the fourth ventricle, measures about 2.5 to 3.0 cm., as reported by Davidoff and Dyke.[3] However, this is a variable figure depending upon the distance at which the exposures are made.

In cases of glioma of the pons, if the ventricular system is well outlined by air, we feel the pneumo-encephalographic appearance is unmistakable. Both of these cases showed a tumefaction of the pons which was well outlined between the floor of the fourth ventricle and the pontine cisterns. These tumors produced a rounded filling defect in the floor of the posterior portion of the third ventricle and a posterior bulging of the floor of the fourth

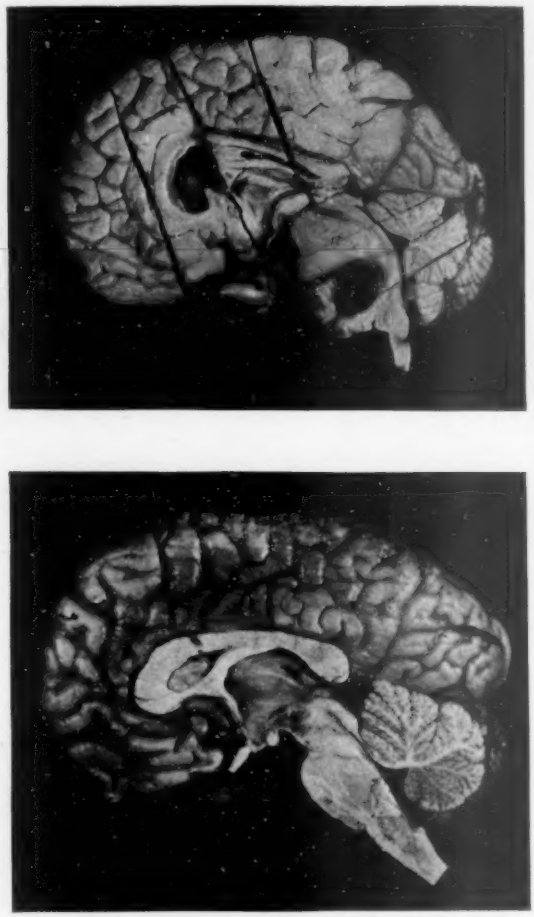

FIG. 4. A comparison of the specimen in Case I (*A*) *above*, with a normal brain (*B*) *below*.

ventricle and aqueduct. There was no air in the subarachnoid space over the cerebrum, and there was flattening or obliteration of the pontine cisterns.

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LESIONS OF THE ACROMIOCLAVICULAR JOINT CAUSING PAIN AND DISABILITY OF THE SHOULDER

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AMONG the various lesions which may induce persistent or recurrent pain and disability of the shoulder, those involving the acromioclavicular joint have received little attention, although they seem to be common.

The acromioclavicular joint has an important function. The range of motion of the arm depends significantly on the mobility of the scapula. The mobility of the scapula depends in turn on the mobility of the shoulder girdle in the sternoclavicular and acromioclavicular joints. These joints render the connection between scapula and sternum sufficiently flexible to permit fairly extensive motion of the scapula along the wall of the chest. Roentgenograms show that there is motion in the acromioclavicular joint during all movements of the arm except pronation and supination (Fig. 1). This means that diminished mobility of the acromioclavicular joint will interfere with most of the movements of the arm in the shoulder.

METHOD

The acromioclavicular joint space is approximately at a right angle to the wing of the scapula. The position of the scapula is variable. In persons with a flat chest and a straight thoracic spine, the shoulder blade is almost vertical and nearly parallel to the frontal plane. The deeper and rounder the thoracic cage, the greater the angle which the scapula forms with the frontal plane; the rounder the curve of the thoracic spine, the greater the deviation of the scapula from the vertical position. The roentgenographic projection is determined, therefore, not by the position of the patient, but by the position of his or her scapula. The central ray should be perpendicular to the wing of the scapula at the level of the acromion. This may be achieved either by placing the patient supine with the shoulder blade of the involved side flat on the cassette or table, the other shoulder being raised accordingly, or by having the patient stand upright with his back to the film and his shoulder drawn backward like a soldier standing at attention. In adipose patients and in the presence of a pathologic or increased physiologic kyphosis, the scapula remains inclined, with its inferior margin pointing backward even in the "attention" position. It is necessary, therefore, to tilt the tube so that the central ray points upward in these cases. The appropriate angle can be determined by palpation of the superior surface of the acromion, the articular surfaces standing usually in the same direction, with the joint space at a right angle to the surface of the acromion. Axial projections, made through the axilla either cephalocaudad or caudocephalad, may give additional information; but their value is somewhat limited as the head of the humerus, parts of the coracoid process, and the glenoid cavity are usually superimposed upon the acromioclavicular joint. Moreover, in the presence of a painful and disabled shoulder, the arm cannot always be abducted sufficiently to obtain a satisfactory projection.

With the exposure factors applied for the ordinary anteroposterior roentgenograms of the shoulder, the acromioclavicular joint usually becomes moderately overexposed or overpenetrated, but not so much as to preclude interpretation, especially if a spot light is used for viewing the films. If this roentgenogram suggests that a lesion of the acromioclavicular joint is responsible for the complaint, additional exposures with a

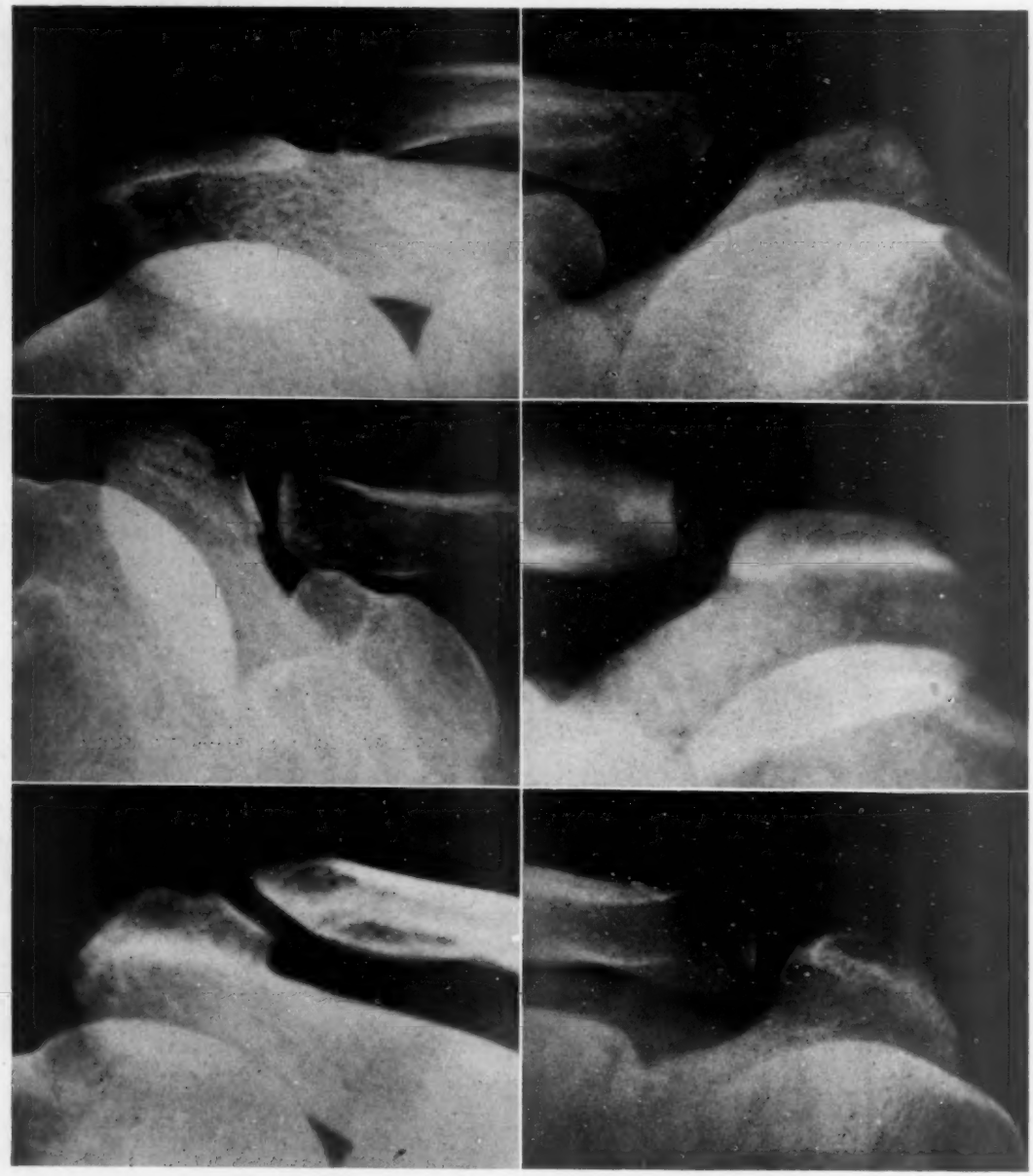

FIG. 1. Normal acromioclavicular joints. Note the variability of outlines, position, and width of joint spaces. Anteroposterior projection, as described in text.

lower kilovoltage may be made. Roentgenograms made at a 36 in. distance, with 30 to 35 kv. and 50 ma-sec., using par-speed screens, are satisfactory. A Potter-Bucky grid need not be used.

NORMAL APPEARANCES

The joint space of the acromioclavicular joint varies between 2 and 5 mm. in width, as measured on the roentgenogram taken at a 36 in. distance, and is wider in the marginal than in the central areas, since the articular surfaces are usually convex, as seen from above. An unusually wide joint space may indicate the presence of an articular disc, which is sometimes found as a normal variation, according to anatomic texts.[5] The articular bony surfaces are

smooth and clean-cut; seen edgewise, they may be straight, notched, concave, or convex, and may differ in shape in the two joints of the same person. The capsule is clearly visible as a cylindric shadow connecting the bony ends and raised very slightly, if at all, above their level in the region of the joint space. The articular bones have a fine mesh-like structure with a very thin cortical layer. Supernumerary ossicles arising from secondary centers of ossification, which are common in the region of the sternoclavicular joint, do not seem to have been observed in the acromioclavicular joint.[7]

The clavicle articulates with the acromion close to the anterior edge of the medial surface of the acromion.

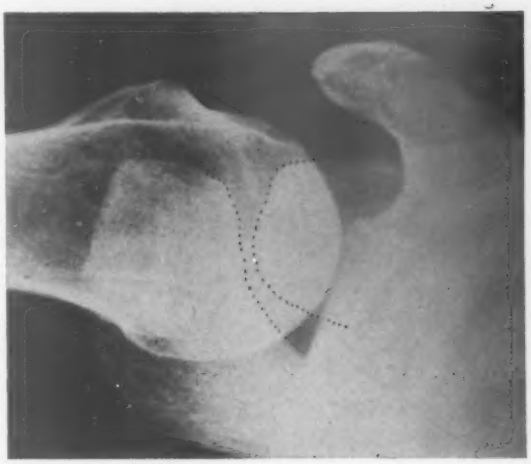

Fig. 2. Normal acromioclavicular joint, cephalocaudal projection upon a curved cassette placed in the axilla. The outlines of the joint, redrawn, are covered by the head of the humerus in this position.

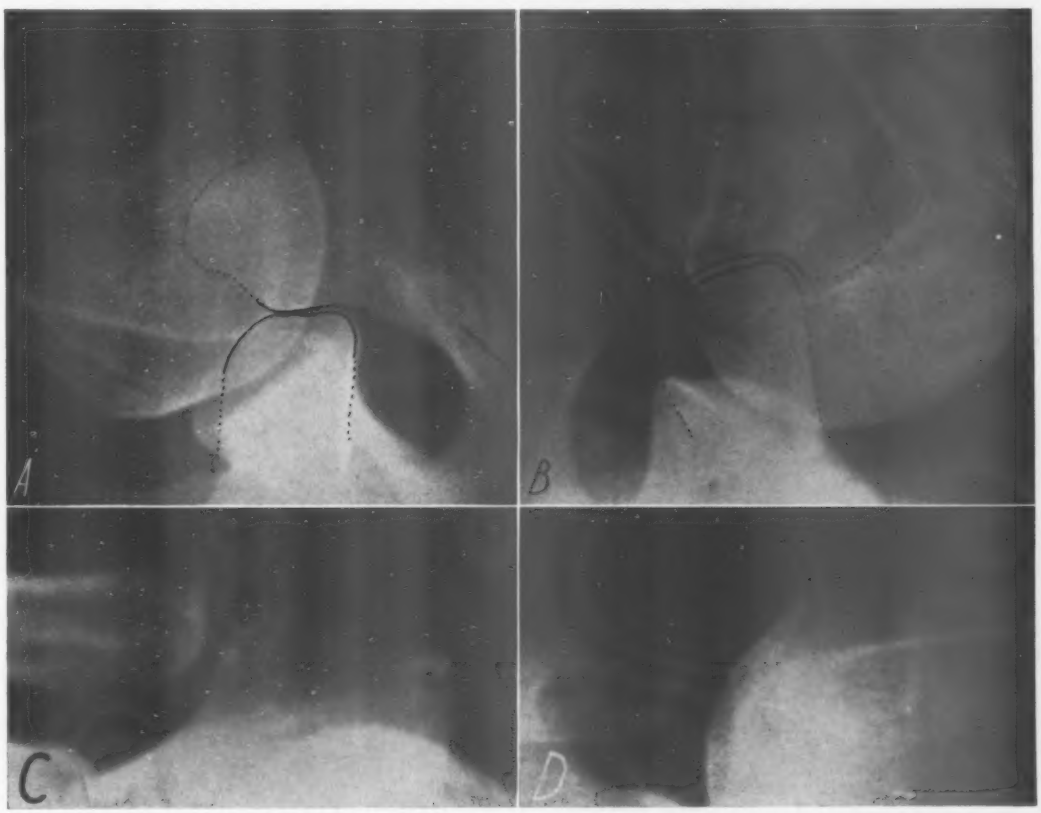

Fig. 3. Position of the articular surfaces during forward (*A*) and backward (*B*) motion of the arm (redrawn), and during normal position (*C*) and elevation (*D*) of the arm.

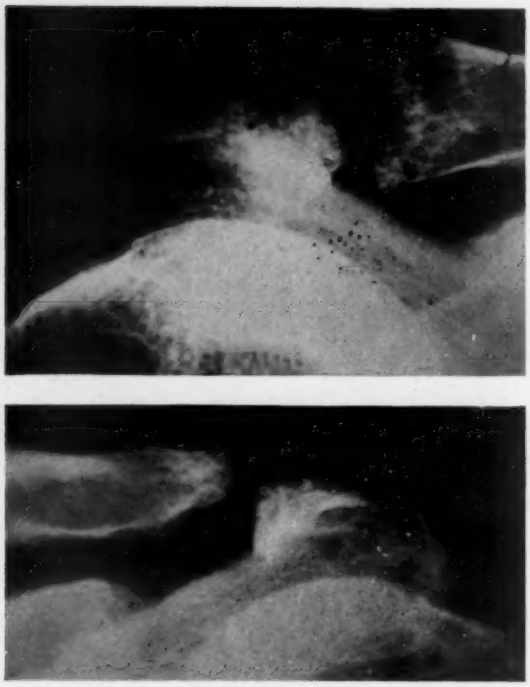

Fig. 4. Changes resulting from recent trauma of the shoulder. Note stippled rarefaction of the bones and fuzzy outlines of articular surfaces.

FRACTURES AND DISLOCATIONS

Fractures of the acromion and dislocations of the acromioclavicular joint are mentioned in most textbooks. They are usually the result of direct trauma. Fractures seem to occur mainly in the lateral part of the acromion, wherefore they rarely involve the joint. As a rule, displacement of fragments is only slight, and the fracture heals readily with a callus formation giving the acromion a dense and rough appearance. Sometimes the fracture line is very difficult to recognize, and a large number of views made in different projections is necessary to demonstrate it. Since the clinical manifestations are usually very mild, it is quite possible that some of these fractures escape recognition. Dislocations of the acromioclavicular joint are less common than fractures, the ratio being 1:4 in our observations. Usually the clavicle is dislocated upward, with or without overriding of the clavicle. Downward dislocation is rare.[1] The coracoacromial and acromioclavicular liga-

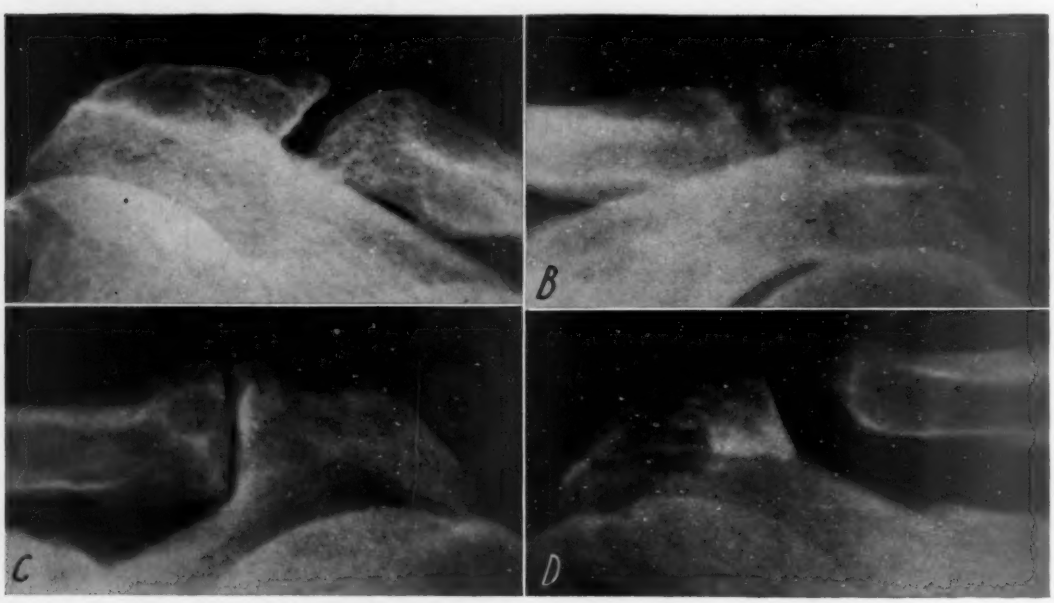

Fig. 5. Hypertrophic (osteo-) arthritis. *A*, normal side; *B*, involved side. *C*, a more advanced case, with severe narrowing of joint space. *D*, another case during an acute exacerbation of symptoms, with widening of joint space possibly caused by effusion.

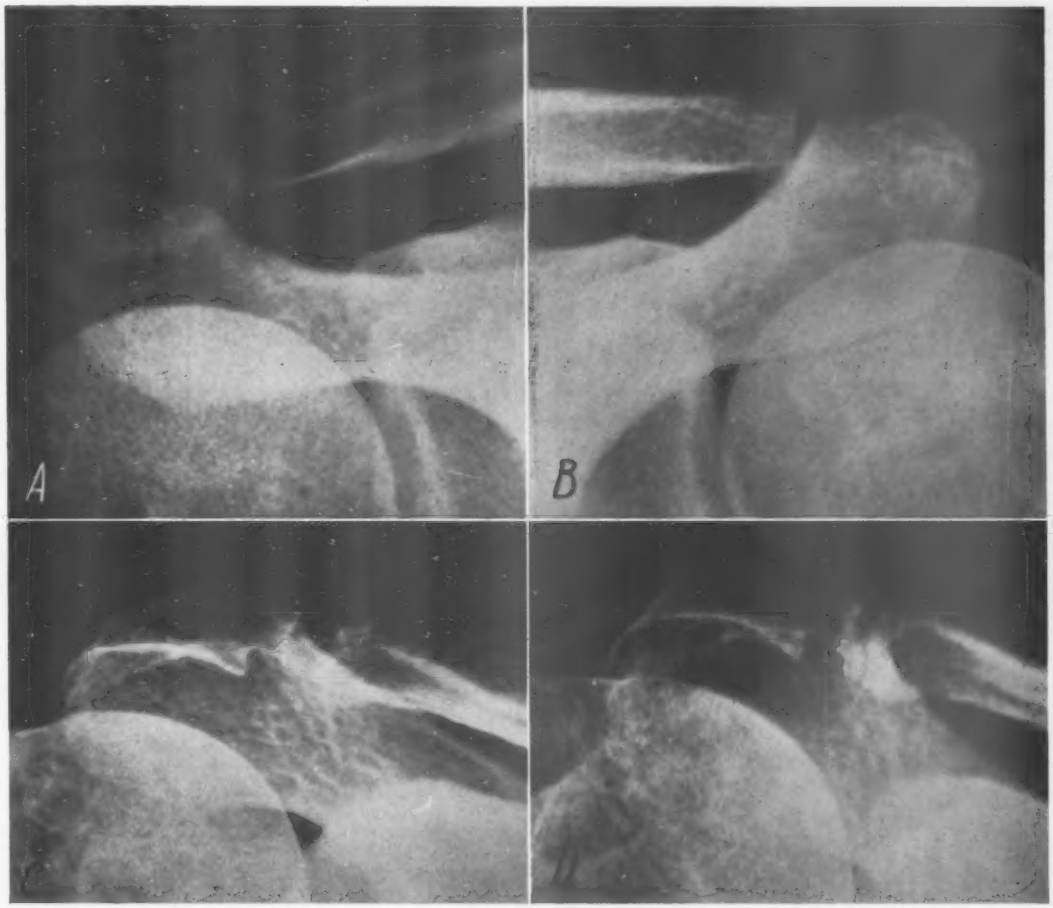

Fig. 6. Atrophic (rheumatoid) arthritis. *A*, normal side; *B*, involved side of a patient with atrophic arthritis of the hands, elbows, and lumbar spine. *C* and *D*, development of ankylosis as a result of rheumatoid arthritis; *C* was taken sixty-four days after *D*.

ments may become injured, and the joint capsule may be torn.

ARTHRITIS

As a synovial or diarthrodial articulation, the acromioclavicular joint may become the seat of any of the various kinds of arthritis. Tuberculous arthritis has been recorded.[4,8] Gonorrheal arthritis leading to complete bony ankylosis was observed in one patient whose roentgenograms are not available. Trophic changes, marked by irregular stippled rarefaction of the articular and para-articular bone, were observed in 2 cases of severe brachial neuritis.

Acute arthritis, clinically indistinguishable from subacromial bursitis, develops occasionally either as a result of sprain, or as a sequela of infection of the nasopharynx. In these cases, the shadow cast by the joint capsule is definitely enlarged (Fig. 5*A*), but there are no other changes seen on roentgenograms. The lesion seems to heal spontaneously in the course of several weeks, and the capsule regains normal size.

Rheumatoid (atrophic) arthritis of the acromioclavicular joint sometimes develops in patients with rheumatoid arthritis of other joints, including those of the spine. Like the peripheral joints, the acromioclavicular joint does not show definite changes on roentgenograms during the in-

Fig. 7. Tumor metastasis. A patient with metastasis of cancer of the breast. *A*, joint normal; *B*, three months later, a small metastasis has lodged close to the articular surface of the clavicle. *C*, five months later, an osteophyte has formed, simulating osteo-arthritis.

cipient and early phases. Later, the capsule becomes enlarged and the articular bone rarefied. The joint space grows narrow, and the articular surfaces may appear eroded. Complete bony ankylosis may develop. In other cases, the lesion becomes arrested before ankylosis occurs, and the articular surfaces with the subchondral bone may then show areas of increased density scattered between areas of rarefaction. Sometimes the articular surfaces become severely eburnated. At this stage of the lesion, rheumatoid arthritis is difficult to distinguish from osteo-arthritis.

Osteo-arthritis (hypertrophic arthritis, degenerative joint disease) seems to develop in the acromioclavicular joint as a result of occupational strain or of repeated trauma. A distinction between "traumatic" and "degenerative" osteo-arthritis is not made here, as the two lesions are anatomically identical. Osteo-arthritis is the most common lesion of the acromioclavicular joint, its incidence being about twice as high as that of all other lesions combined. It produces definite disability of the shoulder. Roentgenograms show the capsule definitely enlarged, usually with moderate widening of the joint space, perhaps as a result of effusion. At more advanced stages, the articular surfaces are eburnated and rough, and their margins may show bony overgrowth; the joint space may then grow narrow. The subchondral bone is usually coarse, the bone trabeculae being increased in thickness but diminished in number. Sometimes small areas of rarefaction are scattered through the area of increased density.

TUMORS

Metastases of cancer of the breast, bronchial carcinoma, and other tumors, are occasionally found lodged very close to the acromioclavicular joint space both in the clavicular and in the sternal articular bones. In 3 of these instances a gradual, apparently reactive eburnation and overgrowth of the articular surfaces was observed in the absence of clinical signs and symptoms. Primary new growth arising from the acromioclavicular joint was not recorded.

CLINICAL MANIFESTATIONS

Dislocations are usually recognized on physical examination, although their recognition is not always easy. Fractures of the acromion are more difficult to diagnose, since pain and disability are very slight in most of these cases, as above mentioned.

Irrespective of its kind and stage, arthritis of the acromioclavicular joint causes pain in the shoulder, often radiating into the arm, wrist, and fingers, with definite limitation of the movements involved in bringing the arm above shoulder level, across the chest, and onto the back. Pronation and supination are not limited, unless pain is so severe as to produce a reflex inhibition; this distinguishes acromioclavicular from humeroscapular arthritis. But the other signs and symptoms are not characteristic, as they may be produced by bursitis, myositis, traumatic lesions of the muscles, tendons, and bones of the shoulder and radicular neuritis caused by disease of the cervical spine. A point of tenderness

confined to the acromioclavicular joint may suggest the presence of a lesion of this joint; but, as a rule, the whole shoulder is tender on palpation and pressure irrespective of the localization of the lesion. Atrophy of a group of muscles may develop, as in the presence of other diseases which induce the "painful shoulder syndrome." The diagnosis is determined by the roentgen findings in a majority of cases.

CONTROL SERIES

In a series of 50 persons without any pain or disability of the shoulder, of various age groups including children and subjects over seventy years of age, calcified subacromial and subdeltoid bursae were present in 3 instances, and various lesions of the cervical spine in 7 cases; the acromioclavicular joints were normal in all of them. Conversely, in a series of 50 cases of recurrent pain and disability of the shoulder, the acromioclavicular joints were found involved alone in 9 patients, and together with flattening of cervical intervertebral discs in 2 instances. This would seem to suggest that the lesions of the acromioclavicular joint here reviewed are actually responsible for the clinical manifestations described, and are not accidental and irrelevant findings.

INCIDENCE

During the past eleven months, when special attention was paid to acromioclavicular lesions, 28 cases were observed. A review of roentgenograms of the shoulder made before that time seems to show that this incidence is the usual one. Acromioclavicular lesions were about eight times as frequent as humeroscapular arthritis; three times as frequent as subdeltoid and subacromial bursitis combined; nearly as frequent as the various traumatic lesions of the muscles and tendons of the shoulder girdle in an industrial and agricultural district; and about one-half as frequent as radicular neuralgia caused by disease of the cervical spine. A study of the age and sex distribution did not disclose any significant findings.

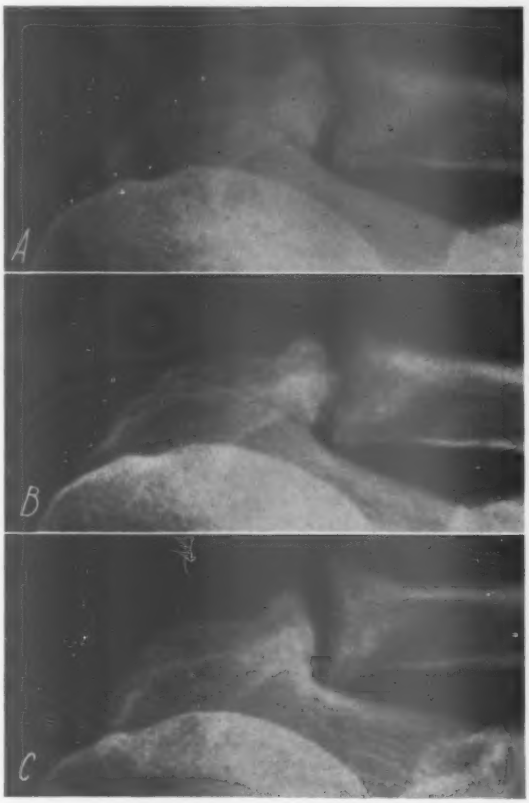

Fig. 8. Result of roentgen therapy. Osteo-arthritis, with narrowing of joint space and marginal overgrowth. *A*, appearance in September, 1942; *B*, May, 1943. No form of treatment was given during the interval. *C*, June, 1943, three weeks after *B* was taken; three treatments with 50 r each were given during the interval. No other therapy was applied.

TREATMENT

All of the patients were referred to this department because various modes of therapy, such as heat applications, analgesics, splints, and injections of a local anesthetic into the deltoid region had failed to produce appreciable or permanent relief. Short wave therapy was ineffective.

Roentgen therapy was applied, using 140 kv., 20 ma., 0.5 mm. Cu plus 1 mm. Al filtration, 50 cm. target-skin distance, with doses of 50 to 70 r (measured in air), through fields 5 cm. square, directed to the acromion from anteriorly or cephalad, at

intervals of four to five days. The results were tested by the method recently described.* In some of the patients pain began to subside rapidly after the second irradiation; in others, six to eight treatments were required to bring about complete or almost complete relief. The movements of the arm in the shoulder usually became normal about ten days after disappearance of the pain; but some slight limitation, generally unnoticed by the patients, tended to persist for several months. A definite decrease of the capsular enlargement was noted on roentgenograms in all instances, and a re-widening of a narrowed joint space, together with decrease of the erosion of the articular surfaces, was seen in 2 cases. Of the 11 patients treated, 5 have been free of symptoms for over six months. No ill effects of this treatment have been recorded.

SUMMARY

1. Pain and limitation of movements of the shoulder are often induced by lesions of the acromioclavicular joint.

2. Osteo-arthritis seems to be the most common lesion which involves the acromioclavicular joint.

3. The clinical manifestations of the various acromioclavicular lesions are virtually identical with those caused by bursitis, myalgia, radicular neuralgia, and traumatic injury of the shoulder girdle.

4. The roentgenographic method and the significant roentgen findings are described.

5. The lesions respond favorably to roentgen therapy with small doses.

* Oppenheimer, A. Development, clinical manifestations, and treatment of rheumatoid arthritis of the apophyseal intervertebral joints. AM. J. ROENTGENOL. & RAD. THERAPY, 1943, *49*, 49–76 (see page 73).

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* I am greatly indebted to Miss Florence E. Turner, Librarian of Boston University School of Medicine, for her assistance in finding these references.

Vol. 51, No. 6

TRAUMATIC SEPARATION OF THE UPPER FEMORAL EPIPHYSIS

A BIRTH INJURY*

By PUTNAM C. KENNEDY, M.D.†
NEW YORK, NEW YORK

TRAUMATIC separation of the upper femoral epiphysis at birth is a rare and interesting lesion. Pfeiffer, in 1936, listed only 15 reported cases, including his own,—12 from the German and 3 from the Italian literature. He was not aware of 3 American cases already published. To these can now be added 3 more but, even including some reports unknown or unavailable to us, the total number would hardly exceed 30. Birth fractures through the diaphyses of clavicle, humerus and femur are common and well known. Separation at birth of the humeral epiphyses and the lower femoral epiphysis is less frequent but has often been described. This is not true, however, of separation of the *upper* femoral epiphysis. In the last ten years neither the Lying-In Hospital nor the Woman's Hospital (both in New York City) have a record of such a birth injury. Moreover, roentgen examination is essential in its diagnosis, as will be apparent in the following case report:

The patient, A. B., a baby girl, was born at term May 16, 1942, in another hospital. A transcript of the record from that institution notes that the position of the fetus was persistently occipitoposterior. Low forceps were applied after thirteen hours of labor. Rotation could not be accomplished and the head was delivered in the posterior position. No difficulty or unusual manipulation is known to have occurred in the extraction of the legs. It is possible, however, that after this operative delivery resuscitation was necessary and the baby's legs may have been abnormally or forcibly handled during this procedure.

Examination of the infant immediately after birth revealed no abnormalities, except of the lower extremities. The thighs were abducted, the knees flexed and the feet deformed (bilateral clubfoot). Each leg was apparently paralyzed and the reflexes were absent. The Wassermann reaction of the mother's blood and the umbilical cord blood was in each case negative.

A copy of the roentgen report dated May 19 states that the bony structure of the lower extremities "is relatively normal in appearance. No dislocation of the hips" and mentions considerable atrophy of the soft tissues. On May 28, twelve days after birth, the report describes "a streak of increased density along the shaft of the left femur in the trochanteric region . . . probably a periosteal reaction."

The infant was admitted to the Orthopedic Service of St. Luke's Hospital on June 4, nineteen days after birth. There was a striking frog-like deformity of the hips, caused by marked flexion, abduction and external rotation. The thighs and hips were swollen, indurated and discolored bluish-red. There was marked limitation of motion at the hip joints and also in the knees and ankles. It was impossible to bring the thighs into a normal position or to extend the legs at the knees. On each side was a talipes equinovarus deformity. Neurological examination revealed normal reflexes in the head and arms but no reflexes in the legs. The skin of the feet and shins seemed unusually shiny, suggesting trophic changes. The baby constantly dribbled urine and this possible indication of a neurological bladder, coupled with the absent reflexes and the skin changes, led to a tentative diagnosis of spastic paraplegia, associated with a spina bifida or spinal birth injury. No manifest spina bifida, however, could be found on physical examination or later by roentgenography. The Kline test was negative.

The diagnosis was clarified on viewing the roentgenograms taken on June 4, the day of admission (Fig. 1). The upper end of each femur was displaced outward and upward from its usual position in relation to the acetabulum. A large mass of heavy callus, quite dense and not fluffy, surrounded the upper end of the right femur. There was a well marked subperiosteal reaction (new bone formation beneath the

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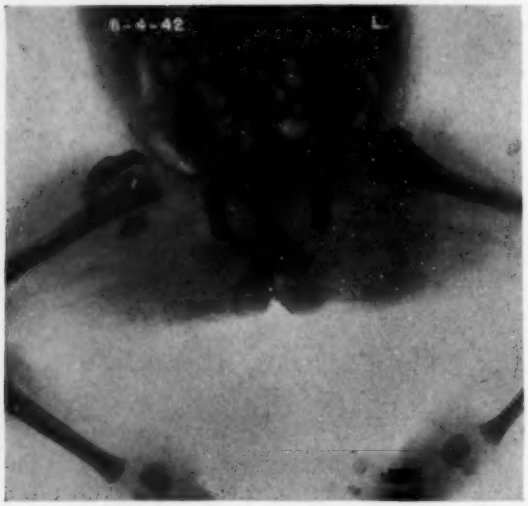

Fig. 1. Roentgenogram taken on admission, nineteen days after birth. Note the heavy callus and prominent subperiosteal reaction around the proximal end of each shaft, which is displaced upward and outward. The hips are held in a frog-like position.

raised-up periosteum) along the proximal two-thirds of the lateral aspect of the femoral shaft, and a fainter, narrower strip along the whole medial margin. The reaction was widest and most prominent in the region of the callus. At the upper end of the left femur was situated a smaller mass of callus, mainly on the upper and lateral aspect. From this a wide, very dense zone of subperiosteal reaction extended down along the lateral edge of the shaft to about the mid-point and apparently continued to the distal end, though visible only along the medial border. No fracture of either femoral diaphysis was seen.

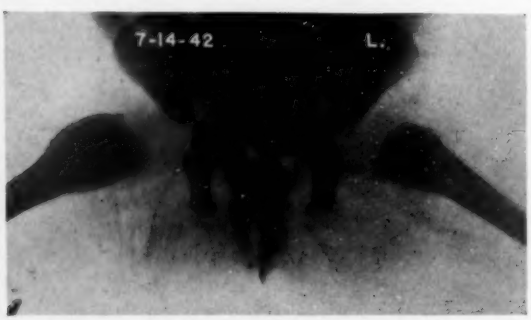

Fig. 2. Roentgenogram two months postpartum. The callus masses have undergone considerable absorption and organization. Note the wide zone of subperiosteal new bone formation along each shaft.

Roentgenograms taken at later dates show progressive organization and absorption of the callus. On July 14, two months postpartum, there is still the frog-like position of the legs (Fig. 2). The space between each acetabulum and the upper end of the femur is still widened. The callus is now organized into new bone and forms a compact, smoothly outlined mass surrounding the proximal end of each diaphysis, the original outlines of which are still discernible within it. A wide zone of dense, laminated, subperiosteal new bone formation extends down the lateral border of each femoral shaft to the distal end.

On August 25, a little over three months post-

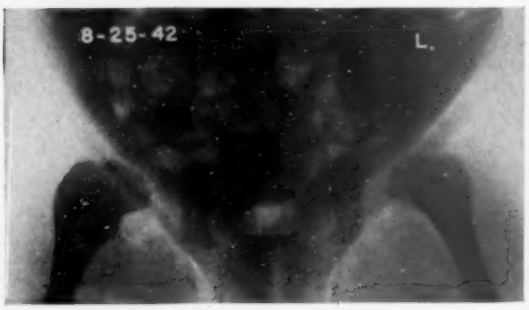

Fig. 3. Roentgenogram about three months postpartum, legs forcibly adducted. Further absorption of callus and subperiosteal new bone; reconstruction of upper end of each femur.

partum, this periosteal reaction is much less prominent (Fig. 3). The club-like enlargement at the proximal end of each diaphysis is smaller and considerably remodeled. In the abducted position the space between acetabulum and femur is still widened; in forcible adduction this distance is not so great. The upper end of the shaft in either position is higher than normal in relation to the Y-shaped fissure of the acetabulum.

On October 9 (Fig. 4 and 5), about five months after birth, the evidences of previous injury are almost gone. The proximal end of each femur, through absorption and remodeling and through the growth process of ossification of the neck, has an appearance more like the normal. Abduction is still maintained and it is impossible to bring the legs together. There is still outward and upward displacement, as will be seen on comparison with roentgenograms of a normal five months old child (Fig. 6). The nucleus of each capital epiphysis is ossified.

During this period corrective measures were

applied to the congenital equinovarus deformities of the feet but no treatment of the hips was instituted.

LITERATURE

The paucity of reports in the literature, especially in American or English journals, prompts a rather complete résumé of them. Pfeiffer states that Pforte[12] described the first case in 1908 but Ehrenfest[3] alludes, without further particulars, to 2 cases reported by Foersterling in 1898. There is also an intriguing will-o'-the-wisp reference in Poland's large monograph, published in 1898: Poland[13] quotes Simpson who

Fig. 4. Roentgenogram about five months postpartum. Hips still flexed, abducted and externally rotated. Remodeling process well advanced. At the proximal end of the right femur the original outlines of the shaft are still visible within the organized callus.

quotes Küstner who quotes Thudichum (1855) as knowing of a case of separation of the upper femoral epiphysis, apparently reported by a man named Bertrandi! Nathan's case[10] and Puppel's[15] case were described as examples of traumatic dislocation of the hip at birth. Harrenstein[6] and Camera[1] independently and almost simultaneously reported their series of observations from which they correctly appraised the true nature of the injury and the resultant lesion. Kleine,[8] in 1933, disproved the contention of Nathan and Puppel and showed that their cases, along with his, were instances of separation of the epiphysis. It remained for Pfeiffer,[11] in 1936, to review and summarize the previous articles, list the cases known to him,

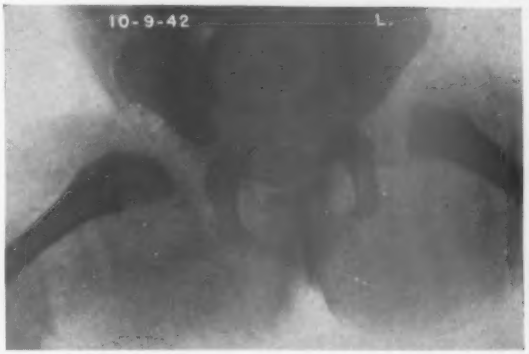

Fig. 5. Roentgenogram about five months postpartum, legs slightly adducted. Note that the proximal end of each femur is still displaced upward and outward. The osseous nucleus of each femoral head is faintly visible and correctly placed in the acetabulum.

and add 3 of his own. Since then Meier[9] has contributed 2 more examples (1939).

In the United States, on the other hand, Truesdell reported a case as early as 1918. This author, who has had a wide experience with birth fractures and epiphyseal injuries, correctly described the lesion and published good roentgenographic studies of it.[19] In 1935, Snedecor, Knapp and Wilson,[17]

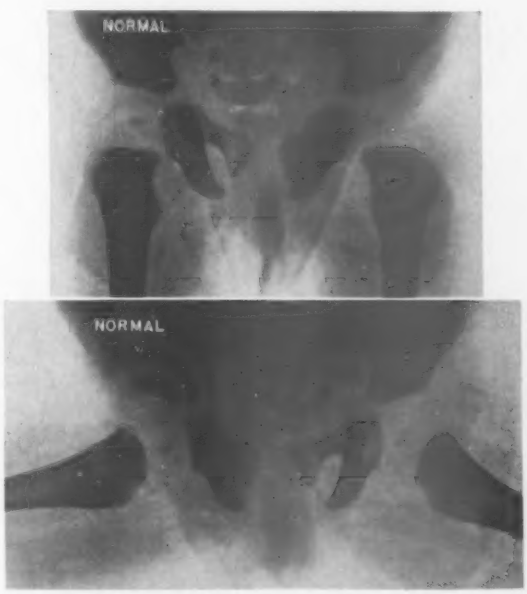

Fig. 6. Roentgenograms of the hips in a normal child aged five months. *Above*, in usual position. *Below*, hips in flexion, abduction and external rotation.

who probably did not have access to Truesdell's paper, reported 2 instances of birth injury to the upper end of the femur to which they gave the name "traumatic ossifying periostitis of the newborn." The roentgenograms of their cases, together with the histories and end-results, show that they really are examples of traumatic separation of the upper femoral epiphysis.

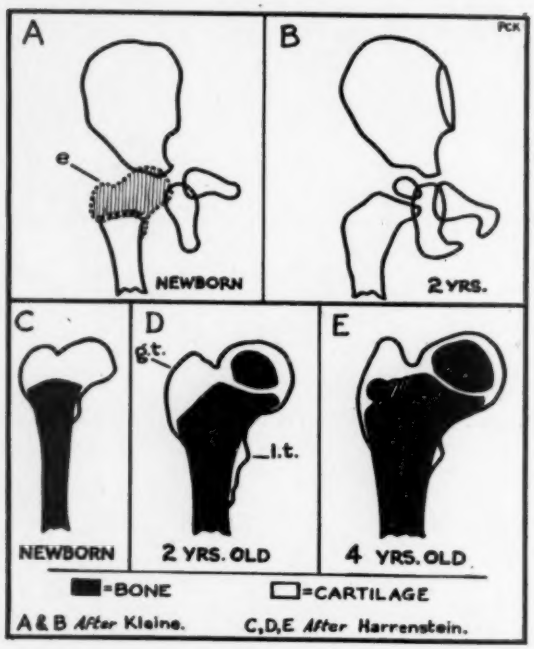

FIG. 7. Diagrams showing development of upper end of femur.

A, at birth. Shaded structure (*e*) is the large cartilaginous upper femoral epiphysis.

B, osseous structures present at the age of two years.

C, at birth the femoral head and both trochanters are included in the cartilaginous epiphysis.

D, at two years of age the head and trochanters (*g.t.* and *l.t.*) are considerably separated through the growth of the femoral neck.

E, at four years, the separation is complete. The head and greater trochanter each have a bony nucleus and separate growth disc.

There are also a few articles[2,14,16] which we have been unable to obtain but their titles suggest additional data on this subject. Two probable cases are listed in Thorndike and Pierce's[18] large chart (H.K.P. and R.G.) but only fragmentary details are given. Undoubtedly several other instances of this injury have been recognized by keen observers but not published. Truesdell,[21] for instance, knows of at least 3 typical cases besides the one in his paper.

Brief accounts of many of the cases in the literature follow (see also Table 1 and Fig. 8):

CASES FROM THE LITERATURE

Truesdell (1918): Breech presentation, delivered by breech extraction. Great difficulty in delivery of shoulders and after-coming head, necessitating vigorous manipulation of lower part of infant's body. At birth, discovery that baby did not move one leg. In a few days, large and firm swelling of thigh. One month later, swelling diminished and function largely restored. Three months after injury, free motion at hip but bony thickening felt in upper thigh. No treatment.

Roentgenograms (Fig. 8, *A*, *B*, *C*): Two weeks postpartum—upper end of femur displaced laterally from acetabulum; large, fluffy callus formation around upper end of shaft. Four weeks postpartum—still lateral displacement; callus denser; subperiosteal new bone formation beneath stripped-up periosteum along proximal two-thirds of shaft. Three months postpartum—still lateral displacement; callus now organized and large mass of new bone surrounding upper end of shaft and region of lesser trochanter; marked subperiosteal new bone formation; ossific nucleus of femoral head already present on affected side but absent on the other; accelerated growth of affected femoral neck.

Nathan (1928) [From Kleine]: Version and breech extraction. During prolonged delivery it was necessary to pull strongly on left leg. At birth, this leg swollen, slightly shortened, externally rotated, abducted, and immobile. First seen by Nathan three and one-half months after injury; roentgenogram at this time showed femoral shaft displaced upward and upper third of shaft bowed outward (varus-like). Treated for eight weeks by "reposition"—plaster immobilization in mid-abduction and strong internal rotation. One and one-half years after birth, position of left femur normal on roentgenograms. Nathan considered this a case of traumatic dislocation of hip but later authors (Kleine, Pfeiffer) disagree with him.

Harrenstein (1929): Case 1. Version and breech extraction. Difficult delivery, including unsuccessful strong traction on right leg. After birth, doughy swelling and no spontaneous movement in right hip; upper femur displaced laterally but leg held in adduction. Pain on passive motion. Three weeks' treatment in a form of extension with vertical traction and occasional lateral pressure on the femoral shaft. After roentgen examination showed improvement treatment was continued for three more weeks. One and

TABLE I

Case	Author	Year of Report	Method of Delivery	Days Postpartum, First Examination	Time Postpartum, Final Examination	End-Result at Time of Last Examination	Treatment
1	Pforte	1908	Version and extraction	3	28 days + autopsy		
2	Truesdell*	1918	Breech extraction	14	3 mo.	Organized callus; free motion at hip	None described
3	Winter	1927	Version	?	Unknown + autopsy		
4	Nathan	1928	Version and extraction	93	20 mo.	Good	"Reposition"; plaster immobilization in mid-abduction and internal rotation
5	Harrenstein, Case I	1929	Version and extraction	17	19 mo.	Good shape and function	Extension, vertical traction + lateral pressure on shaft
6	Harrenstein, Case II	1929	Version and extraction	17	11 mo.	Coxa vara	None
7	Camera, Case I	1930	Version?—breech extraction	2–3	2 yr.		
8	Camera, Case II	1930	Version?—breech extraction	2–3	2 yr.	Severe coxa vara	
9	Camera, Case III	1930	Version?—breech extraction	17	Unknown		
10	Puppel	1930	Version and extraction	5	9 mo.	Return to normal	Abduction
11	Kleine	1933	Version and extraction	1	7 mo.	Severe coxa vara	Supporting-fixation bandage; abduction, slight internal rotation
12	Med. Clinic	1935	Version and extraction	5	Unknown		
13	Snedecor,* Case I	1935	Version and extraction	40	4 mo.	Callus organized	None
14	Snedecor,* Case II	1935	Version and extraction	5	24 days	Large callus	None
15	Pfeiffer, Case I	1936	Version and extraction	19	4 wk. + autopsy		None
16	Pfeiffer, Case II	1936	Version and extraction	3	14 mo.	Return to normal; no coxa vara	Lorenz primary position
17	Pfeiffer, Case III	1936	Version and extraction	42	7 mo.	No coxa vara	Tibial extension, traction
18	Epstein and Klein	1936	Version and extraction	?	?		
19	Meier,* Case I	1939	Breech presentation and extraction	10	1 yr.	No coxa vara	Extension; 90° flexion at hip; internal rotation
20	Meier,* Case II	1939	Version and extraction	?30	8 wk. + autopsy	Mild coxa vara	None
21	Kennedy*	1944	Head presentation low forceps	19	5 mo.	Poor; still abducted and externally rotated	None yet

* New cases added to Pfeiffer's list.

one-half years after injury, shape and motion of hip normal.

Roentgenograms: Seventeen days postpartum—right femoral shaft displaced upward and outward; upper limit of diaphysis less sharply defined than on opposite side; fluffy callus on medial aspect of upper end of femur. Five weeks postpartum—less displacement; callus compact and spread out over upper end. One and one-half years postpartum—position good; ossific nucleus for femoral head larger than on uninjured side; cartilaginous disc between head and neck narrower than on uninjured side.

Case II. Version and breech extraction. Difficult delivery, with unsuccessful strong traction on right leg in order to disengage foot wedged between fetal head and maternal pelvis. At birth, hip swollen and immobile, continuing thus for four to five weeks. Expectant treatment at home for various reasons. Partial return of active motion at end of seven weeks but full range not till eleven months (even then, slightly limited extension).

Roentgenograms (Fig. 8, *D*, *E*): Seventeen days postpartum—upward and lateral displacement of proximal end of femoral shaft; upper limit of diaphysis fuzzy and surrounded by callus. Seven weeks postpartum—callus increased but compact; subperiosteal new bone formation along upper third of shaft. Eight months postpartum—femoral head nucleus visible on affected side but not on other. Eleven months postpartum—still slight lateral displacement; head nucleus larger and growth disc narrower on affected side; femoral neck and medial border of upper shaft curved on injured side, indicating coxa vara deformity; diaphysis shortened 0.5 cm.

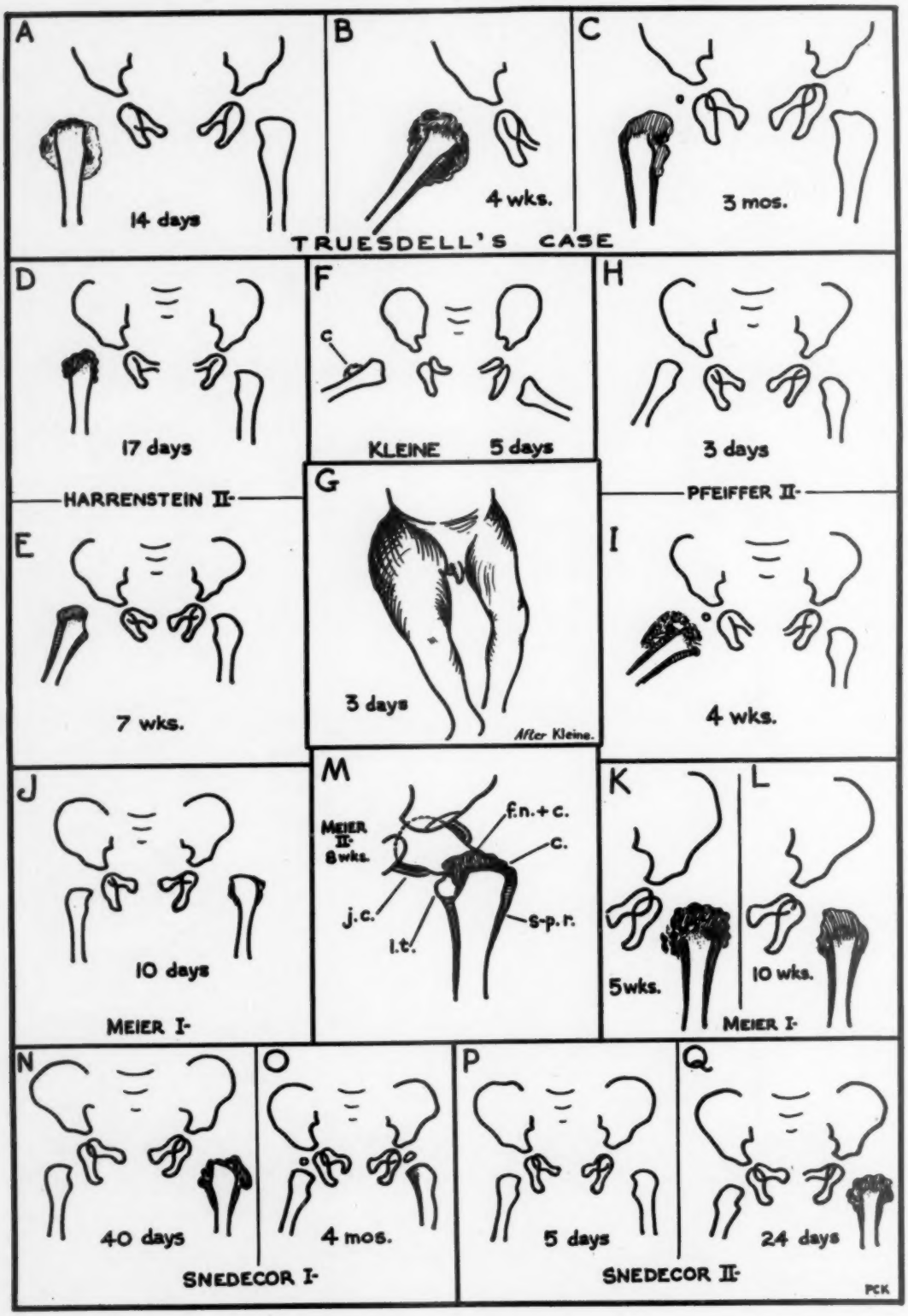

FIG. 8. See next page for description.

Puppel (1930) [From Kleine]: Version and breech extraction. Traction on right foot. On fourth day after birth, swelling, shortening, external rotation, and limited motion of right thigh. Treatment in abduction. Incomplete serial roentgen studies.

Roentgenograms: nine days postpartum—upper end of right femur higher than left. Six weeks postpartum—still high; faint, fan-like callus at upper end of right femur. Ten months postpartum—full restoration to normal. Puppel considered this a case of traumatic dislocation of the hip but later authors (Kleine, Pfeiffer) disagree with him.

Kleine (1933): Version and breech extraction, necessitated by placenta previa in thirty-fourth week of pregnancy. Traction on right foot. On third day, right thigh swollen and painful; right leg shortened and externally rotated. Treated for four weeks with Pappschien's supporting-fixation bandage in abduction and slight internal rotation. Active motion possible after one month and full motion after three months.

Roentgenograms (Fig. 8, *F*): Five days postpartum—upper end of right femur displaced upwards and outwards; slight amount of callus. Nineteen days postpartum—large callus mass around upper end of femur; subperiosteal reaction along proximal third of shaft. One month postpartum—callus increased in size, density and compactness. Three months postpartum—callus absorbed and organized. Six months postpartum—slight elevation, bowing and widening of upper end, indicating traumatic coxa vara deformity; ossific nucleus of head present on affected side. Seven months postpartum—coxa vara; advanced growth on injured side, with larger head nucleus, narrower growth disc and increased width in region of trochanters.

Snedecor et al. (1935): Case I. Version and breech extraction. Forty days after birth, pain and limited motion noticed in thigh. No treatment.

Roentgenograms (Fig. 8, *N*, *O*): Forty days postpartum—callus around upper end of femoral diaphysis; subperiosteal new bone formation along upper fourth of shaft. Four months postpartum—callus absorbed and organized and upper end of femur remodeled; traces of injury still present.

Case II. Version and breech extraction. Soon after birth, left leg swollen, discolored and painful. No treatment. Home follow-up stated that baby recovered completely.

Roentgenograms (Fig. 8, *P*, *Q*): Five days postpartum—no displacement or callus. Twenty-four days postpartum—large callus around upper end of left femur; subperiosteal reaction around proximal fourth of shaft.

Pfeiffer (1936): Case I. Version and breech extraction. Right upper arm fractured at birth. Later parents noticed swelling of left hip. Eighteen days after birth, marked swelling, loss of active motion, painful passive motion. Planned treatment not carried out because baby soon died of pneumonia.

Roentgenogram: About eighteen days postpartum—upward and outward displacement of upper end of femoral shaft; callus mass surrounding it.

Case II. Version and breech extraction. Immediately after birth, right thigh swollen, discolored, crepitant, externally rotated, and immobile. On sixteenth day postpartum, swelling diminished and plaster cast applied in the Lorenz primary position (for about two weeks). Two months after injury examination showed free motion in affected hip.

Roentgenograms (Fig. 8, *H*, *I*): Three days postpartum—upper end of right femur displaced outwards and upwards; no callus visible. Sixteen days postpartum—beginning callus formation. Four weeks postpartum—still outward displacement; large callus around proximal end of diaphysis; prominent subperiosteal reaction along upper third of shaft; ossific nucleus of femoral head already discernible on injured side. Fourteen months postpartum—ossific head epiphysis larger on injured side than on the other; head epiphysis correctly placed in acetabulum, both sides; no coxa vara deformity.

Case III. Version and breech extraction. Referring physician had diagnosed "dislocation-fracture" and applied cast. First seen by Pfeiffer six weeks postpartum; right thigh then swollen and right leg visibly shortened. Plaster cast with right hip in abduction and flexion (Lorenz primary position) tried but deemed unsatisfactory as promoting development of coxa vara. Then longitudinal tibial traction until displacement corrected, followed by cast with hip in abduction. About four and one-half months after birth, active motion when cast removed. At seven months hip appeared normal but still slight limitation of abduction.

FIG. 8. Diagrams of several cases from the literature.

A, *B*, and *C*, evolution of callus, at first flaky and finally compact and organized.

D and *E*, marked displacement of proximal end of diaphysis.

F, very early appearance of callus (*c*), five days after birth.

G, after Kleine's photograph of baby three days following birth injury.

H and *I*, premature ossification of capital epiphysis on injured side, as early as four weeks after birth.

J, *K* and *L*, characteristic stages of lesion.

M, diagram of Meier's autopsy specimen: *c*, callus; *f.n.+c.*, osseous growth of femoral neck plus callus; *s-p.r.*, subperiosteal new bone formation; *j.c.*, intact joint capsule; *l.t.*, avulsed cartilage of lesser trochanter.

N, *O*, *P* and *Q*, Snedecor's 2 cases, called "traumatic ossifying periostitis" but showing characteristics of other cases of epiphyseal separation.

Roentgenograms: About six weeks postpartum—moderate abduction, proximal end of femur displaced upwards and outwards; small amount of callus. About eight weeks postpartum—displaced higher; in Lorenz position; apparent severe coxa vara; more callus visible. About five and one-half months postpartum—proximal end of femur widened in region of trochanters; ossific head epiphysis already present on affected side and correctly placed in acetabulum. Seven months postpartum—normal position of head and shaft on each side; no apparent coxa vara deformity; head epiphysis larger and cartilaginous growth disc narrower on injured side; still slight thickening of upper end of femur in trochanteric region.

Meier (1939): Case 1. Breech (footling) presentation and extraction. Traction on left foot because umbilical cord caught between legs. Delivery not difficult nor prolonged, however. Four days after birth, swelling and limited motion of left thigh. Nine days after birth, swelling, abduction, external rotation, and slight crepitus on passive motion. Treatment—form of extension with 90° flexion at hip and internal rotation. At four months of age no limitation of motion, at eight months patient stood alone, and at one year patient walked alone, without functional impairment.

Roentgenograms (Fig. 8, *J*, *K*, *L*): Ten days postpartum—upper end of left femur displaced upwards and outwards and externally rotated; on close inspection, faint callus formation and periosteal reaction could be seen. Five weeks postpartum—large callus mass around proximal end of femur; wide zone of subperiosteal reaction along medial and lateral borders of upper three-fourths of shaft. Ten weeks postpartum—callus denser and more compact; subperiosteal new bone formation advanced. One year postpartum—upper end of left femur thickened; head epiphysis larger and growth disc narrower on injured side; increased growth in lesser trochanteric region on injured side and possible ossification of lesser trochanter nucleus.

Case II. Version and breech extraction. Premature birth, transverse position, prolapsed cord. Weight at birth: 1800 grams. Painful swelling of right hip detected four weeks after delivery; also external rotation and 1 cm. shortening of right leg. No treatment. Infant died at eight weeks of erysipelas. Roentgenogram similar to first case but changes less extensive.

Autopsy findings: Femoral diaphysis externally rotated almost 90° in relation to the neck. Upper femoral epiphysis slipped medially on the top curved surface of diaphysis. Angle between neck and shaft narrowed 4 to 5°. Callus at proximal end of diaphysis. No changes in actual head cartilage. Joint capsule and ligamentum teres intact. No increased joint fluid or old hemorrhage into hip joint.

ANATOMY AND PATHOLOGY

At birth the upper femoral epiphysis is a single large mass of cartilage surmounting the slightly convex upper end of the shaft (Fig. 7). From it are derived the head and both trochanters. The osseous nucleus of the femoral head does not appear till the fifth to seventh month of life (Hodges). The nucleus of the greater trochanter does not begin to ossify until the third or fourth year. In the meantime growth of the femoral neck proceeds steadily, lengthening the distance between head and greater trochanter until in a four year old child, they are completely separated (Fig. 7, *C*, *D*, *E*). At this time there is a definite growth disc of cartilage between each of these bony epiphyses and the neck. The growth of the neck also separates the head from the cartilage of the lesser trochanter. The hip joint capsule is attached to the femoral neck beyond the head; at birth the attachment is proximal to the junction between bony diaphysis and the upper cartilaginous epiphysis. In early years the periosteum over this junction is very tough and strong.[13]

When violence is exerted at birth on the upper end of the femur the cartilaginous mass as a whole is displaced medially and downward off the curved upper surface of the diaphysis. The periosteum is stripped up for a variable distance down along the shaft, sometimes as far as the distal end. There is hemorrhage at the site of epiphyseal separation and beneath the torn-up periosteum. The joint capsule and ligamentum teres remain intact, however, and the cartilaginous femoral head is lodged in the acetabulum. No dislocation of the hip joint is therefore present (Fig. 8, *M*).

Reparative processes begin immediately, marked by the swift production of bony callus. This callus is usually abundant and forms a large, irregular mass around the proximal end of the diaphysis and the base of the displaced epiphysis. There is a larger amount, as a rule, on the upper outer aspect than on the inner. A great deal of the callus (especially in severe cases) lies outside the raised periosteum. Beneath the latter, new bone formation takes place rapidly. The next steps are the absorption and organization of the callus and subperiosteal osseous tissue. The club-like mass gradually disap-

pears and the femoral shaft and neck are reconstructed and remodeled, aided by the osseous growth of the neck proceeding regularly upward from the diaphysis. As a matter of fact, the growth of the head and neck in such an injured femur is accelerated, temporarily at least, and it is common for the nucleus of the head to ossify prematurely. When the bony nucleus of the opposite capital epiphysis appears, it is seen to be obviously smaller than its fellow on the injured side, and the cartilage space or growth disc between nucleus and neck on the sound side is considerably wider.

Part of the above knowledge has been obtained from study of roentgenograms and part from examination of specimens dissected at autopsy. Meier, for instance, described the pathological changes present in an infant dying of erysipelas eight weeks after the birth injury (Fig. 8, *M*). He found the diaphysis of the affected femur externally rotated almost 90° in relation to the neck. The upper cartilaginous epiphysis had slipped medially on the top curved surface of the diaphysis, narrowing the angle between neck and shaft by about 5°. There was bony callus at the proximal end of the shaft. No changes were present in the cartilage of the head itself and the joint capsule and ligamentum teres were intact. There was no evidence of increased joint fluid or of previous hemorrhage into the joint cavity. The cartilaginous lesser trochanter had apparently been avulsed along with the periosteum.

Harrenstein had previously demonstrated the results of this injury produced experimentally in dead newborn infants. The cartilaginous upper epiphysis was separated, all in one piece, at its junction with the diaphysis. The periosteum was separated from the cortex; the joint capsule was intact and not perforated.

CAUSE OF INJURY

All writers are agreed that an unusually forcible manipulation of the infant's leg is the cause of the injury. This is also the case in birth fractures of the femoral shaft or separation of the lower femoral epiphysis. In roughly half the cases of shaft fractures delivery has been by breech extraction, often following version, though some have occurred during cesarean section or "normal" delivery. Among the mechanisms cited by Truesdell[20] are: (1) in the course of podalic version, injury to the femur *in utero* while trying to pull down a leg; (2) torsion of the leg in the vagina (footling); (3) injury while extricating the infant during a difficult cesarean section; (4) abnormal positions and strains on the leg or legs through too vigorous manipulation during difficult extraction of the after-coming head, and (5) even injury after delivery while swinging the child by the leg to promote respiration.

On the other hand, *every case but one* of separation of the upper femoral epiphysis reviewed herein occurred during breech presentation and extraction or version and breech extraction. Although some of the case reports mention no particular difficulty or maneuver, several others note a "difficult" or "prolonged" delivery and "strong" or "powerful" traction on one of the legs. In one, the lower part of the infant's body was manipulated forcibly in trying to deliver the shoulders. In another, a foot was wedged between fetal head and maternal pelvis. The humerus of one baby was also fractured. Unequal traction was used in one instance because the umbilical cord was caught between the legs. In 2 cases there may have been an element of haste—one with placenta previa and one (premature) with a prolapsed cord.

In our case, the lone exception, there was vertex presentation and occipitoposterior position. Difficulty was encountered in delivering the head but none, as far as we can find, in extracting the legs. It is still possible that the legs were forcibly manipulated during attempts to resuscitate the child, if such were necessary.

Harrenstein, in his experiments on dead newborn infants, could not produce epiphyseal separation by traction alone. He was successful in one trial by using strong traction and at the same time forcing the femoral head against the taut iliofemoral

ligament; in another, by combining traction with moderate rotation; and in a third, by using traction plus abduction and rotatory movements. Fixation of the pelvis was a prerequisite. Gurlt's experiments are cited by Ehrenfest and Meier as showing that the epiphyseal separation could be produced only by strong traction together with rotation or abduction.

CLINICAL CHARACTERISTICS

The obstetrician is usually unaware at the moment that the limb has been injured. He can often hear the "crack" when a bony shaft is broken but no audible sound attends the separation of the epiphysis. Pforte, it is true, is said to have heard a "thud" but his case is the exception. The obstetrician in Nathan's case had a "feeling that something was broken" and Harrenstein, in his experiments, "felt something give way."

In almost every instance, however, the injury is not apparent till the delivery is completed. Then it will be noticed that the baby does not move one leg. Swelling of the thigh may or may not be present so soon. If the first is overlooked and the second is slight or absent, the injury may go undetected for a few days but by that time many signs are present, loudly proclaiming it. These are: (1) marked, firm swelling of the affected hip and thigh; (2) slight but noticeable shortening of that leg; (3) no active motion at the hip; (4) pain on attempts at passive motion; and (5) external rotation. Also present sometimes are: (6) bluish-red discoloration of the thigh; (7) crepitus with passive movements; (8)abduction or (9) adduction. The shortening, external rotation and abduction have occasionally been noticed at the time of birth.

The swelling gradually subsides, lasting from two weeks to two months. In its place a "bony thickening" can be felt in the upper thigh, caused by the callus mass around the upper end of the shaft. After about a month partial active motion is usually resumed and after two to five months free motion. The latter, however, may be postponed in the severer cases.

ROENTGENOGRAPHIC CHARACTERISTICS

The first roentgenographic sign is *displacement of the upper margin of the femoral diaphysis*, always outward and usually always upward. This can be ascertained by comparison with the structures of the other side and by noting the position of the proximal end of the shaft in relation to the Y-shaped fissure of the acetabulum. In a few severe cases the end of the shaft is seen above the upper limit of the Y-fissure, lateral to the lower portion of the ilium, but usually it lies below or at the level of the fissure. In most instances the diaphysis is externally rotated; it may be either abducted or adducted.

Faint *callus* shadows can be discovered during the second week, though in one instance (Kleine's case, Fig. 8, *F*) as early as the fifth day. After the first two weeks the callus is readily visible, usually abundant and forming a club-like mass around the proximal end of the diaphysis. At first it is flaky or cloudy but later (fourth week and following) it becomes more compact, more dense and more heavily calcified.

At about the same time as the callus the characteristic sign of *subperiosteal new bone formation*, a result of the avulsion of the periosteum from the cortex, makes its appearance. This extends distally from the upper end of the shaft for variable distances, often beyond the mid-point and occasionally to the lower end. As the callus becomes denser, so does the subperiosteal tissue. The upper limits of this periosteal reaction can often be seen, incorporated in the mass of callus.

During the second and third months the callus and subperiosteal new bone undergo *organization and absorption*. The club-like mass becomes progressively smaller, its edges smoother and its internal structure trabeculated. Reconstruction and remodeling of the femoral neck and upper diaphysis are apparent in the roentgenograms. There may still be outward and sometimes upward displacement of the diaphysis, dependent on the severity of the injury and the subsequent treatment.

Another characteristic sign is the *pre-*

mature ossification of the capital epiphysis on the injured side. This appeared as early as the twenty-eighth day in one case (Pfeiffer, Case II, Fig. 8, *I*) and at three months in another (Truesdell, Fig. 8, *C*). After the opposite nucleus is ossified, it is seen in the roentgenograms to be smaller than the one on the injured side, as late as one to one and one-half years after birth (Fig. 10). The cartilaginous growth disc likewise is narrower on the affected side, more evidence of accelerated growth in that region. When the bony nucleus of the injured femur becomes visible it is seen to be correctly lodged in the acetabulum.

At later examinations there is usually only widening of the proximal end of the diaphysis in the trochanteric region; traces of the old callus and the remodeling process may still be present. There is often a coxa vara deformity, with narrowing of the neck-shaft angle; the upper third or fourth of the shaft may be bowed in a varus manner. In some instances—the least severe cases—the bones appear normal.

PROGNOSIS AND TREATMENT

Lesser degrees of injury are usually followed by good end-results. This may be true without any treatment (Snedecor's first case) or with only moderate treatment (Puppel, Harrenstein, Case 1; Pfeiffer, Case II). Severer injury often results in a coxa vara deformity, especially if no treatment has been given (Harrenstein, Case II; Meier, Case II). Moderate treatment in Kleine's case did not forestall the development of a considerable coxa vara; rather extensive measures in 2 other cases (Pfeiffer, Case III; Meier, Case I) did prevent this deformity.

Pfeiffer discusses the prognosis at length. He separates the cases into two groups: (1) less severe injury, with good prognosis; and (2) severe injury, with poorer prognosis. In the *first* group (Fig. 9, *A*, *B*) roentgenograms show only a moderate outward and slight upward displacement of the proximal end of the diaphysis. Presumably the perichondro-periosteal membrane has not been ruptured but only lifted up (a point put forward by Harrenstein), thus preventing any great displacement of the fragments. These are the cases with good end-results despite no treatment or only gentle supportive measures.

In the *second* group (Fig. 9, *C*, *D*) roentgenograms show a greater deviation outward and a marked displacement upward.

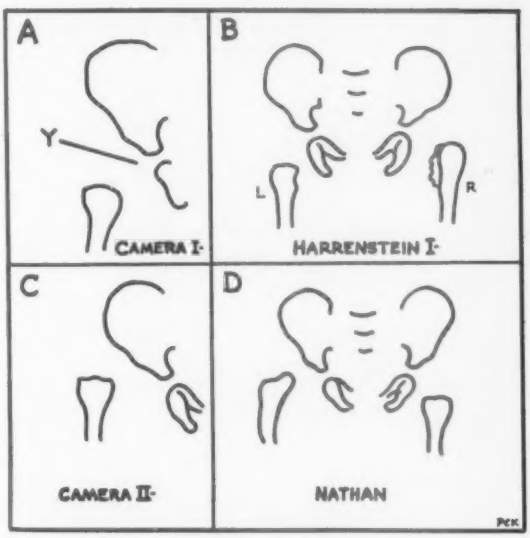

FIG. 9. Diagrams after Pfeiffer.

A and *B*, mild injury and good prognosis. The proximal end of the femoral diaphysis lies below the Y-shaped fissure of the acetabulum (*Y*).

C and *D*, severe injury and poorer prognosis. The proximal end of the diaphysis is above or on a level with the Y-fissure.

The upper end of the diaphysis is found above the Y-fissure and sometimes even above the upper rim of the acetabulum. The soft tissue injury here must have been very great, associated with tearing of the periosteum and severe damage to the surrounding muscles. In these cases coxa vara is almost certain to develop unless prompt and thorough orthopedic treatment is instituted. Pfeiffer cites his own third case, Camera's second, and Kleine's single case as illustrations of the second group (Fig. 10).

Our own case falls into this second class with respect to the degree of displacement and apparent severity of injury; we cannot tell yet whether coxa vara deformities are

developing, though they are likely to be. Treatment of the hips has just begun (five months after birth).

The orthopedic procedures described in the literature have not been uniform (see Table 1 and case reports).

DISCUSSION

Perhaps the diagnosis of traumatic separation of the upper femoral epiphysis will be made more often in the future. This will come about if the obstetrician examines the infant more closely after podalic version and breech extraction and if serial roentgenograms are taken in suspected cases for at least three weeks after birth. In the first week the roentgenologist should detect the displacement of the upper end of the diaphysis. The next roentgenograms, a week or two later, will show the early callus and confirm his tentative diagnosis.

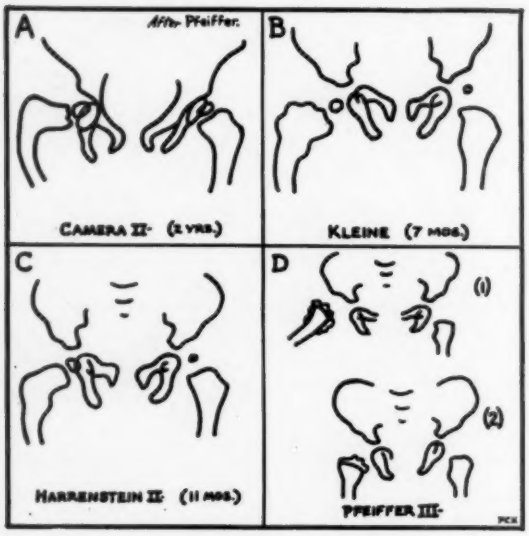

FIG. 10. Diagrams illustrating end-results.

A, *B*, and *C*, varying degrees of coxa vara. Note the larger capital epiphysis and narrower growth disc on the affected side.

D, good result following orthopedic treatment. (1) Unsatisfactory position after trial in Lorenz primary position. (2) Good position after extension and tibial traction.

It is just as important to follow the later course of the lesion by serial roentgenograms. As the callus increases and then is absorbed and organized, the reconstruction of the femoral neck and upper shaft can be observed and appraised. The development of a coxa vara deformity can be foreseen and appropriate measures taken to avert it. Further, the effects of treatment can be noted and evaluated.

Serial roentgenograms will also rule out the remote possibility of a traumatic dislocation of the hip occurring at birth. Such a lesion has long been the subject of controversy, most authors denying its existence. Nathan and Puppel, mentioned above, considered their cases examples of traumatic dislocation. One of Kleine's cogent reasons why Nathan's case was really an epiphyseal separation is that Nathan did not see the patient until three and one-half months after birth, when the callus was all gone and the roentgenogram showed only upward displacement and bowing of the proximal end of the diaphysis. Likewise, Kleine's objection to Puppel's diagnosis is that there were no roentgenograms between the ninth and forty-second days. On the former date the right femur was displaced upward—the only sign; on the latter a faint fan-like callus was present (though apparently missed by Puppel). Roentgenograms in the interval would probably have shown the characteristic development of the callus, a phenomenon not found in dislocation of the hip.

Observation of later roentgenograms provides another argument against dislocation of the hip. When the nucleus of the femoral head ossifies it is seen to be in its normal position in the acetabulum. Moreover, as Kleine points out, osseous growth of the affected capital epiphysis and femoral neck are accelerated in these cases of epiphyseal separation; on the contrary, hypoplasia is the rule in dislocation of the hip joint.

SUMMARY

(1) Traumatic separation of the upper femoral epiphysis is a rare birth injury. Twenty cases from the literature (mostly foreign) have been reviewed and our own case report added.

(2) Every case except one has occurred during podalic version and breech extraction or breech presentation and extraction. Our case is exceptional in that the position was occipitoposterior, delivery by low forceps, and that no known violence was exerted on the legs. It is also the only case in which the lesion is bilateral.

(3) The upper femoral epiphysis at birth is a single cartilaginous mass, including head, neck and both trochanters. It is displaced medially and downward at its junction with the diaphysis. The periosteum is usually stripped up and sometimes ruptured. The joint capsule and ligamentum teres remain intact.

(4) Rotatory movements combined with traction on the leg are necessary to produce the injury.

(5) The clinical signs are swelling, slight shortening, limitation of active motion, painful passive motion, and external rotation; also sometimes discoloration, crepitus, and abduction or adduction.

(6) The roentgenographic signs are displacement of the proximal end of the diaphysis (upward and outward); rapid, profuse callus formation and prominent subperiosteal reaction around the upper end of the shaft; absorption of callus and reconstruction of the femoral neck; and premature ossification of the capital epiphysis and accelerated growth of the femoral neck on the injured side.

(7) The prognosis is good in cases of milder injury but rather poor when the initial trauma and displacement have been severe. Coxa vara is a frequent end-result in the latter group of cases.

(8) The value of serial roentgenograms is emphasized in the diagnosis and following of these cases.

The author wishes to express his appreciation to Dr. Charles W. Breimer for his help and advice during the writing of this paper.

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AN OSCILLATING PLEURAL HERNIA*

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A PLEURAL hernia shifting through the mediastinum is not a strange or unknown entity to those specialists doing many pneumothoraces, or a large amount of roentgenography. A reversible pleural hernia, which swings pendulum like across the median line, from right to left, is however an exceedingly interesting phenomenon.

A thorough search through modern literature reveals only 11 cases of transmediastinal hernia reported. To this list we wish to add another which is believed to be the twelfth case of oscillating pleural hernia recorded. The first mediastinal hernia was reported by Cantieri[2] in 1925. Subsequently, single cases have been reported by Bordet and Parodi,[1] Locatelli,[6] Raimondi and Sangiovanni,[7] Timpano,[9] Rey, Rey and Pucci,[8] Dufourt, Muller and Jandaud,[3] Levi-Valensi and Charles;[5] Finkelstein[4] reported 3 cases.

There are two sites where these hernias are likely to occur. One is located in the superior mediastinum and anteriorly. In this area the air pressure pushes the pleura to the opposite side directly behind the sternum. The second location of transmediastinal hernias is usually in the posterior mediastinum, inferiorly. Here the hernia occurs between the esophagus and the arch of the aorta.

REPORT OF CASE

R. B., white, male, aged thirty-four.

Family History. Father and mother dead; ages and causes unknown. Two brothers and one sister living and well. One brother died in infancy. Wife living and well. No cancer, tuberculosis, neuropsychiatric or cardiac disease known in any members of family.

Personal History. Measles, pertussis and smallpox during childhood. No illness in early adult life. Denies syphilis. Gonorrhea in 1919. No recurrence or residuals. Smokes cigarettes moderately. Denies the use of alcohol or drugs. Has had no operation. Fractured right elbow at age of five or six.

Military History. Enlisted in Army June 2, 1918; discharged May 19, 1919. Not overseas. Hospitalized two weeks early in 1919, with tonsillitis, and cough. No tonsillectomy.

Present Illness. Patient states that in the latter part of 1920 he began to have a cough, soreness throughout the chest; fever for a few days at a time, occurring about twice a year, generally in the spring or fall. Was hospitalized two or three weeks in 1923; two weeks in 1929, and from September 13, 1933, until November 15, 1934. Was sick four months at home in 1925. Began feeling tired all the time. Soreness in the chest; night sweats; loss of weight—lost 21 lb. in the past six or seven months; coughs some all the day but more at night. Expectoration is moderate. No frank hemorrhage. Sputum contains small particles of blood occasionally. Has had sore throat at times—none in the past two or three years.

Worked in the coal mines in 1920 and continued to 1929. Worked as an attendant in a hospital from 1929 to September, 1933. He lost time for two or three weeks; once for five months in 1920 because of illness.

Sputum positive for tubercle bacilli in 1923.

Chief Complaints. Fatigability, cough and expectoration, dyspnea, loss of weight, anorexia, vomiting, night sweats.

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Physical Examination.

General appearance: Inspection reveals a fairly well nourished and developed white male, who looks chronically ill. Height 68¼ inches; weight 133½ lb.; average 155–160 lb.

Scalp: Hair normal; no baldness.

Skull: No elevation, or depressions.

Eyes: Right, vision—20/50; corrective lenses —20/20; pupil reacts to light and accommodation. Left, vision—20/50; corrective lenses 20/20; pupil reacts to light and accommodation.

Ears: Right, conversation voice—2 feet; whispered voice—0; drum membrane—retracted, adherent. Left conversation voice—35 ft.; whispered voice—35 feet; Weber test—equal; Rinne, right—negative for all forks, no tones heard, left positive, all tones heard.

Nose: Negative. Breathing space ample.

Face: A few small scars and bluish pigmentation on face, due to coal mine injury five years ago.

Mouth: Mucosa normal.

Teeth: All uppers extracted and most of lowers extracted.

Tongue: Normal.

Tonsils: Small.

Neck: Thyroid not enlarged.

Chest: Medium length, width and thickness; there is slight tenderness on pressure, right base, posteriorly. Mobility fair and equal.

Palpation: Fremitus: right lung—slight increase throughout; left lung—not increased.

Percussion: right lung—impaired resonance above 3rd rib and 7th dorsal spine. Left lung—impaired resonance above 2nd rib and 5th dorsal spine.

Auscultation: Right and left lungs—whispered voice sounds not increased. Breath sounds vesicular. No râles heard.

Gastrointestinal tract: Patient gives a history of constipation extending over a period of several years past. States that the stools are frequently hard and frequently one stool in several days. States that different kinds of remedies have been used regularly about once or twice a week for the relief of constipation over a period of the past three years. No history of vomiting. No hematemesis. No jaundice. Jaundice is not present at this time. States that there is an occasional discomfort in the abdomen, and that this discomfort is general. This discomfort does not bear any relation to meals, according to patient's statement.

Wassermann and Kahn reactions, negative. Erythrocyte count, 4,600,000; leukocyte count, 13,800, with 80 per cent polymorphonuclears; hemoglobin 85 per cent. Urinalysis, negative.

Genitourinary report: The kidneys and bladder region are negative. Genitals, negative. There is no adenopathy. The urine is clear in both portions, and essentially negative for cellular elements. The prostate and vesicles are normal. The prostatic and expressed fluid is negative.

The patient was studied by the surgical collapse board. The right artificial pneumothorax was discontinued, as it was thought the lung had been collapsed an adequate length of time. A pneumothorax was started in the left because of more recent lesion. It was discontinued because of repeated spontaneous pneumothoraces.

Patient has been on bed rest, as he is of the nervous type with multiple complaints, although he is not difficult to handle. The left lung has been completely re-expanded. The heart is retracted to the right, but the right lung is not entirely re-expanded, as shown on the roentgenograms. Pain in the right chest, apparently due to high negative pressure, is relieved by instillation of small amount of air. On examination, the left side is essentially clear. Last sputum examination was positive. Gaffky III.

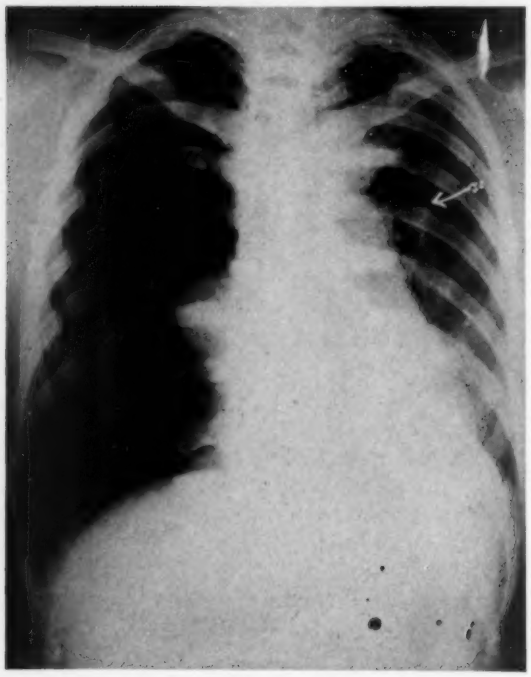
Roentgen Examination.

Gastrointestinal tract: Roentgenoscopy—Opaque meal passed the esophagus without hesitation. Stomach filled normally. Both curvatures are regular in outline and freely movable, exhibiting no filling defects, or deformities. The duodenal bulb filled, showing no evidence of abnormality, spilled over to the right and formed a normal first and second portion of the duodenum.

The roentgenographic examination of the gastrointestinal tract confirms roentgenoscopic findings, showing on the immediate films a well filled stomach and duodenal bulb. The colon is normal. The gallbladder is well visualized, displaying no alteration in appearance to suggest cholecystic disease.

Roentgen diagnosis: Gastrointestinal tract normal; gallbladder normal.

Chest: The roentgenographic examination of the chest at 2 meter distance shows the bony framework to be barrel shaped and symmetrical, exhibiting no kyphosis, cervical, or forked regions.



Right Left

FIG. 1

Domes of both diaphragms are smooth. Costophrenic angles are deep, acute and clear, displaying no evidence of fluid, adhesions, or thickened pleura.

The heart image is of normal size and shape and position for the type chest possessing it. Borders are regular; measurements are not accentuated, and there is no roentgen evidence of organic heart disease.

The lobes of the right chest are totally compressed by a pneumothorax. The right dome of the diaphragm is depressed. The heart, thoracic and mediastinal contents are also displaced to the left. A semicircular shadow line is seen extending from the region of the first anterior rib outward in a graceful curve and ending in the region of the upper border of the third anterior rib, attributed to a pleural hernia (Fig. 1).

The lobes of the left chest show slight accentuation of the bronchial tree markings, with interstitial fibrosis in the right upper. The vertebral first and second interspace trunks are prominent, fuzzy, beaded and interweaving, and apparently reaching the periphery, attributed to an inflammatory process. This snow storm effect mimics the findings of tuberculosis.

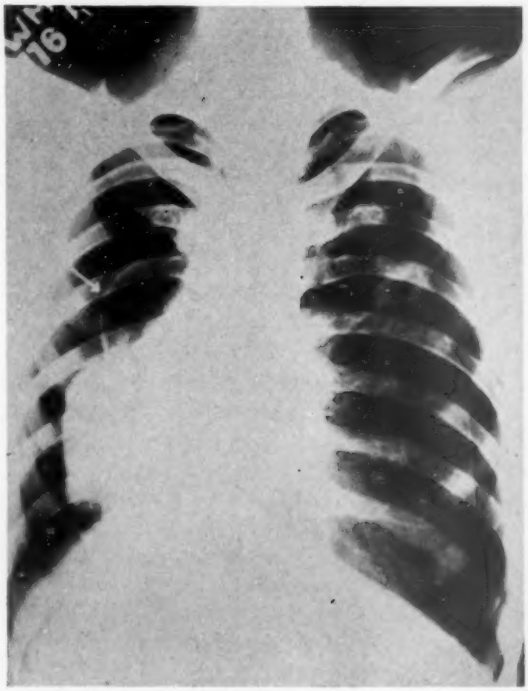
The examination of the chest roentgenogram taken two years later shows a total collapse of the lobes of the right chest and also a snow storm density infiltrating the left upper lobe and apex. A pneumothorax in seen in left chest extending from the apex to the base and occupying the peripheral lung zone, producing approximately 25 per cent collapse, lobes of left chest.

A semicircular shadow line is seen in the right upper chest extending from the region of the first anterior rib outward in a graceful bulging shadow line and ending in the region of the upper border of the third anterior rib, attributed to a pleural hernia (Fig. 2).

The pleural hernia, seen on previous roentgenogram, in the left root lung zone has now reversed itself to the root lung zone of the right hemithorax.

Evaluating the roentgenographic findings displayed in this interesting phenomenon, wherein the pleural hernia of the left chest is observed, and demonstrated to shift to the right hemithorax, this is believed to be caused by the incarcerated air in the right pneumothorax cavity having been absorbed, thereby creating a large vacuum or an extensive negative pressure area.

The small artificial pneumothorax occupying the peripheral lung zone of the left chest was



Right Left

FIG. 2

therefore sufficient pressure to displace the pleural hernia to the opposite side, thereby causing this exceedingly interesting phenomenon.

The authors are indebted to Parvin M. Eaton for his aid in preparing the illustrations accompanying this article.

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A SIMPLIFIED METHOD OF BRONCHOGRAPHY

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SEVERAL standard procedures for the instillation of iodized oil into the bronchial tree are in general use but all methods have certain drawbacks and do not adapt themselves well to the needs of a military roentgen-ray department. One of the early methods advocated was to introduce the opaque oil via the cricothyroid membrane, having previously anesthetized the skin locally with 0.5 per cent novocaine intradermally.[1] The preliminary preparation for this method is time consuming and requires careful aseptic technique; in addition, there is a constant danger that the oil will escape into the fascial planes of the neck, and even gravitate into the retrosternal tissues, where it remains for years. Dysphonia, edema of the glottis, dysphagia and subcutaneous emphysema are constant hazards with this technique.[2]

In some institutions it is the practice to allow the otolaryngologist to introduce the iodized oil, either via the bronchoscope or directly into the larynx, before sending the patient to the department. This method seems to be inadequate, for as a rule the roentgenograms disclose a very limited visualization of one segment of the bronchial tree, and in most cases sufficient time has elapsed before the roentgenograms are taken to permit the oil to find its way into the alveoli. When the latter happens, no matter what method is used, accurate interpretation is out of the question. Clinically unsuspected lesions, therefore, are overlooked because of the segmental visualization.

The most universally employed routine is the transglottic method using a curved metal cannula. The preliminary preparation requires complete anesthesia of the pharynx and larynx with 5 per cent cocaine and adrenalin. During the introduction of the oil it is necessary for the patient to grasp the tongue firmly with one hand, enhancing the grip with gauze. Theoretically, this opens the glottis automatically and allows the oil to collect in the larynx where it is aspirated slowly and flows into the bronchial tree by gravity.

The several disadvantages of this method can be summarized as follows: There is a certain element of danger in the employment of cocaine but, if inadequate amounts are used, the anesthesia is not complete enough to prevent the occurrence of the gagging reflex. This reflex is very easily touched off in the darkened roentgenoscopic room where the operator, having his attention directed to the flow of the oil into the proper channels, may allow the cannula to deviate to the sensitive pharynx. Furthermore, to obtain a complete visualization of the bronchial tree it is necessary to tilt the patient in several positions and in so doing the situation becomes progressively more awkward, for during the entire operation it is essential that the tongue remain immobolized and that the curved cannula, through which the oil is being injected, remain in place.

The simple method proposed here was evolved by necessity over a period of months. It requires no preliminary preparation of the patient and no elaborate equipment and is therefore particularly well adapted to Army use. The only medication is ½ to 1 gr. of codeine administered about an hour before the patient arrives at the roentgen department, to diminish the cough reflex. After the patient has entered the roentgenoscopic room, a preliminary explanation is given him, describing carefully and simply what is to be done and what is expected of him in the way of cooperation. Two admonitions are stressed repeatedly: that he must not cough during the entire procedure and that he must not swallow, no matter how strong the desire becomes. If the impulse either to cough or to swallow

seems overwhelming, it can be overcome, he is told, by increasing the respiratory rate. It is expressed graphically to the patient by saying "pant like a dog." If this instruction is followed it becomes mechanically impossible for the patient to allow either reflex to gain ascendency over this rapid voluntary respiration. The forward position of the tongue during panting accomplishes the opening of the glottis just as well as holding of the tongue by the patient.

The necessity for a perfect understanding between the subject and the roentgenoscopist cannot be stressed too strongly. Where the patient is not cooperative or does not grasp the import of the instructions immediately, it becomes necessary to restate them forcibly and frequently.

The next step is to introduce a 14 F. sterilized catheter through the nose and along its floor. By gentle manipulation under roentgenoscopic control, the catheter passes easily along the posterior wall of the nasopharynx to a point just above the larynx. A large syringe is filled with 20 cc. of the iodized oil which has been previously warmed to body temperature, and to this syringe is attached the free end of the catheter. The injection of the oil is started and in most cases the first few cubic centimeters will fill both pyriform sinuses and cascade gradually into the larynx. The progress of the iodized oil is noted roentgenoscopically and a constant check is kept both on the amount of oil in the pharynx and the amount flowing to the lower branch bronchi. If one side seems to fill more rapidly than the other, which is usually the case, the patient can be tilted to the opposite side. The flexibility of the catheter and the freedom of the patient from encumbrance by equipment permits extreme changes in position.

When about 15 cc. has been instilled and a large quantity is observed flowing down the trachea, the table is tilted quickly into the horizontal position, allowing gravity to carry the opaque medium into the posterior branches of the lower bronchi. The patient is next shifted into the semi-erect position and the remaining 5 cc. of the oil is injected. Immediately thereafter the table is reversed into the Trendelenburg position to fill the upper branches. During the time the patient is in the supine position he is asked to turn first to the right and then to the left, until finally all branches of the bronchi are filled.

The patient is now given permission to swallow and expectorate at will but is instructed to refrain from coughing until after the roentgenograms are made. All efforts are directed towards decreasing the time interval between roentgenoscopy and the taking of the roentgenograms to prevent the oil from gravitating into the terminal bronchioles and alveoli.

Posteroanterior and both oblique views are taken routinely in all bronchographic examinations. It is necessary to employ a lateral projection occasionally and the need for this position can be determined during roentgenoscopy. The technique for chest roentgenography is maintained except that 8 to 10 kv. (peak) is added to allow the visualization of those branches otherwise obscured by the heart shadow. Following the taking of the bronchograms, the patient is instructed to cough and to lower the head over the side of the bed several times a day in order to drain out as much as possible of the iodized oil. This serves two purposes: it prevents a reaction in those individuals sensitive to iodine, and it decreases the amount of iodized oil seen in subsequent roentgen examinations of the chest.

This method, after some practice, rarely takes more than fifteen to twenty minutes from beginning to end. Occasionally, a noncooperative patient will require considerably longer but, in a large series of cases, complete failure is rare, once the routine has been mastered. It is often impossible to visualize the entire bronchial tree, although an effort to do so is strongly recommended. At times bronchiectasis will be so far advanced that most of the oil will be collected in the diseased portion of the lungs. Once

the diagnosis has been established in such cases, however, an attempt at further filling is unnecessary, unless precise knowledge of the extent of the lesion for surgical evaluation is required.

CONCLUSIONS

A simple, effective, rapid method is described to accomplish bronchography. It is completely adequate and does not require either an elaborate set-up, specialized skill, or protracted preparation.

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Vol. 51, No. 6

THE USE OF A MODIFIED OPAQUE BARIUM SULFATE MIXTURE IN ROENTGENOGRAPHY OF THE COLON*

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THE purpose of this paper is to describe the use of a modified opaque barium sulfate mixture in roentgenography of the colon. This method permits simultaneous demonstration of (1) the luminal contour as available from the ordinary, densely opaque barium sulfate enema, and (2) a view of the normally distended colonic lumen and its contents, if any. In the ordinary method a view of the lumen is not possible, due to its opacification. After evacuation of the modified opaque mixture it is possible to obtain a view of the rugae such as seen after the evacuation in the ordinary barium enema method.

Following success with the use of a modified opaque meal in roentgenography of the gastrointestinal tract,[1] attention was directed to the colon where, after experimentation with various mixtures, it was found that a mixture containing 75 grams of barium sulfate and 5 grams of gum acacia to 2 quarts of water was the most suitable. In this manner sufficient contrast is maintained and, at the same time, the colon assumes a "pearly" or "porcelain" semitransparency which is enough to visualize any abnormal or normal luminal contents. Ordinarily, it would require the instillation of gas or air, with or without a small amount of barium sulfate, to visualize the colon in this state of normal distention.

The method is especially valuable for the study of the colon in the regions of the flexures and sigmoid and in those cases with marked colonic redundancy. One is thus able to resolve the component parts of various loops which are ordinarily clumped and densely opacified. This eliminates any additional oblique views and minimizes the number of lesions in the flexures and sigmoid which may occasionally be overlooked due to superimposition of a normally opacified portion of the colon. Polyps low in the colon are more easily seen, and by their persistence in size, shape and position are differentiated from fecal masses. In the presence of a sigmoid mass it may occasionally be difficult to differentiate between cancer or an inflammatory mass associated with diverticula, or to determine the presence of both conditions. With the modified mixture the diverticula will be more likely to fill.

Roentgenoscopy is more difficult than under ordinary circumstances. This, however, is somewhat balanced by utilizing the full accommodation of the roentgenoscopist before beginning the examination and by the actual roentgenograms. Besides, experience has shown that this thin mixture passes in much more rapidly, so that the total roentgenoscopy time for the examination is reduced. Thus, it may be permissible to increase the milliamperage to a slight extent.

Another finding has been the visualization of numerous fecal masses in cases which would otherwise appear well prepared with the routine method. This proves that very few individuals, even after thorough preparation, are completely evacuated, and that in the ordinary method a coating of barium sulfate sufficiently opacifies and blends the fecal masses with the barium sulfate in the lumen.

In cases of obstruction a small amount of this modified mixture is more likely to get by the narrowed portion to outline the lesion than is the ordinary thicker mixture, for too often the thick barium sulfate mixture halts at the most distal point of ob-

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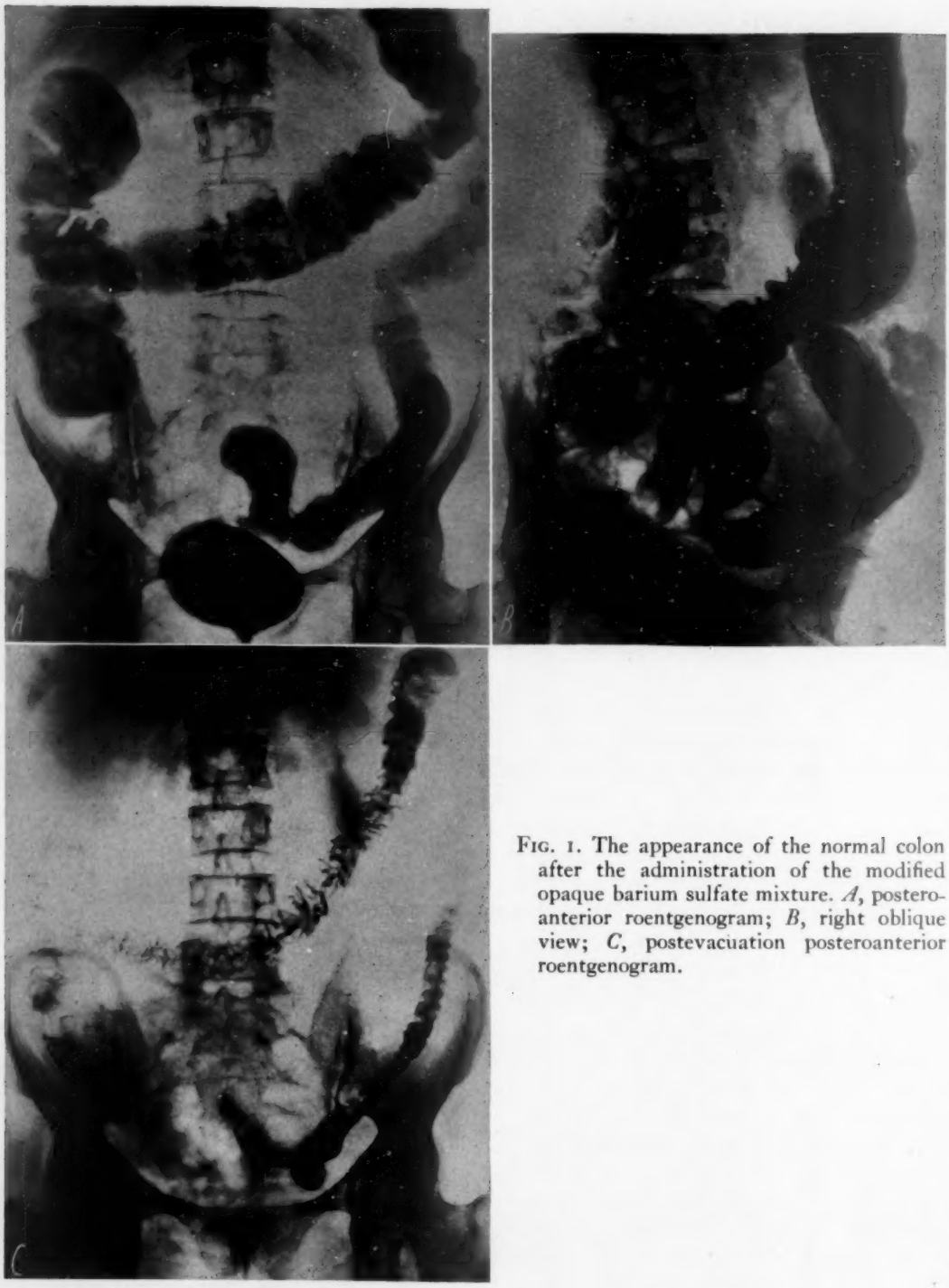

FIG. 1. The appearance of the normal colon after the administration of the modified opaque barium sulfate mixture. *A*, posteroanterior roentgenogram; *B*, right oblique view; *C*, postevacuation posteroanterior roentgenogram.

struction and one can only state that there is an obstruction, and nothing else. Further, it is our distinct impression that patients seem to hold more of this thin mixture before complaining of discomfort.

Correlation between roentgen and pathological studies has usually shown that the lesion pathologically is much larger than has actually been seen on the roentgenogram, despite the distortion factor which

Fig. 2. Right oblique view of barium enema with ordinary barium mixture, showing a mass near the hepatic flexure.

Fig. 3. Same case as Figure 2; roentgenogram made the following day, using the modified opaque barium sulfate mixture. Note the difference in the size of the mass.

actually tends to increase the size of the lesion with the technique ordinarily used for barium enemas. The modified method has resulted in demonstrating a larger lesion than the thicker mixture in the same case during the same day or within twenty-four to forty-eight hours. This is due to the fact that the thick barium sulfate opacifies and thus obscures the edges of the lesion by passing either superiorly, inferiorly, anteriorly or posteriorly to it. This does not obtain in the modified mixture and thus the results more nearly approximate the actual pathological picture.

Occasionally upon the completion of a barium enema with the ordinary method the examiner feels perplexed, and upon reexamination finds no additional information to enable him to reach a conclusion. In such a case it might be advisable to make the enema examination again, with the modified mixture. This different approach may be productive of sufficient information to enable the examiner to arrive at a conclusion.

CONCLUSION

The use of a modified opaque barium sulfate mixture in roentgenography of the colon has been described.

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THE TREATMENT OF ACCESSIBLE MALIGNANT TUMORS WITH SHORT DISTANCE LOW VOLTAGE ROENTGEN RAYS*

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AN advance was made in the method of treatment of accessible malignant tumors by the introduction of modern apparatus for short distance low voltage roentgen therapy. This advance originated with the work of Schaefer, Witte, Chaoul and Adam. Working at first with cathode rays and starting with the assumption that

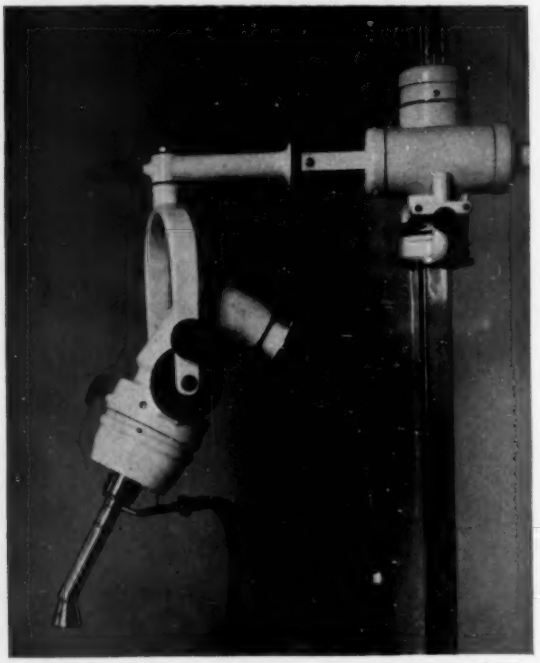


FIG. 1

the success previously obtained with radium was not due to the quality of the radiation but to the distribution of the energy absorption in the tissues, they evolved a method of roentgen treatment which gives a similar distribution. This was achieved by the use of a short focal-skin distance and a low voltage, the object being to confine the radiation effect to a small volume of tissue.

Apparatus. The apparatus (Fig. 1) was made by Messrs. Siemens and was originally designed by Schaefer and Witte for intracavitary roentgen therapy. Its use in the treatment of skin and other accessible tumors was developed by Chaoul and Adam. This tube (Fig. 2) is constructed so that the stream of cathode rays passes down an earthed metal cylinder to strike the target situated at the far end. The roentgen rays used in treatment pass through the anode which is water cooled. In this way the source of the roentgen rays can be brought to within a few millimeters of the lesion to be treated.

More recently Messrs. Philips have constructed an apparatus for short distance roentgen treatment (Fig. 3). The Philips

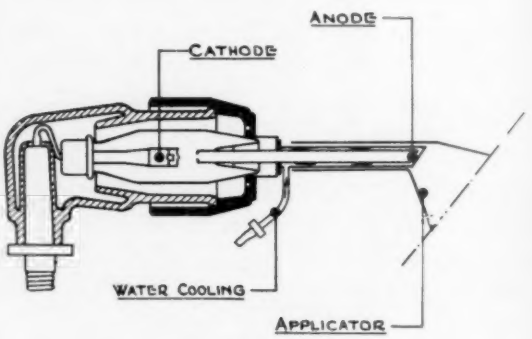


FIG. 2

roentgen tube (Fig. 4) is designed so as to avoid a transmission target and thus reduce the inherent filtration. In this tube the cathode is earthed and the stream of cathode rays passes in the opposite direction to the roentgen rays utilized. The anode is separated from the lesion by a glass window and a "philite" cap and is cooled by radiation to the walls of the tube

* Prepared for presentation with a color film at the International Cancer Congress, Atlantic City, N. J., September, 1939.

which are themselves air-cooled. The inherent filtration of the Siemens tube is equivalent to 6.3 mm. Al and of the Philips tube to 0.2 mm. Al.

Advantages. The main advantages that short distance low voltage roentgen treatment has over the use of radium are the ease of application, the fact that the radiation may be limited to the tumor and its immediate vicinity (the rest of the patient is not irradiated at all), the high intensity of the radiation, the low initial cost of the apparatus, and the freedom from danger to the personnel of the department. The high intensity is of particular importance for it allows large numbers of patients to be treated in a short time by limiting individual treatment times to a matter of a few minutes. Short treatment times also mean a minimum of disturbance with the patients' usual occupations and save the discomfort accompanying prolonged applications to certain parts of the body.

Limitations. This method of treatment is strictly limited in its application to lesions that are directly accessible either

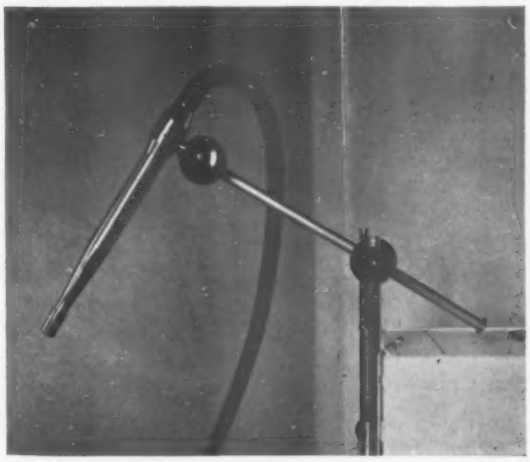


Fig. 3

by virtue of their situation or by means of previous surgical exposure, and are of limited depth. These limitations result from the distribution of the radiation and are an integral part of the design of the treatment.

Method of "Caustic" Roentgen Therapy. The majority of tumors that can be brought into direct contact with an applicator involve the skin or mucous membranes so that consideration of skin tolerance, that important limiting factor in so many roentgen treatments, does not arise. Overdosage in short distance therapy is not often followed by such serious consequences as it is in high voltage treatment for it is con-

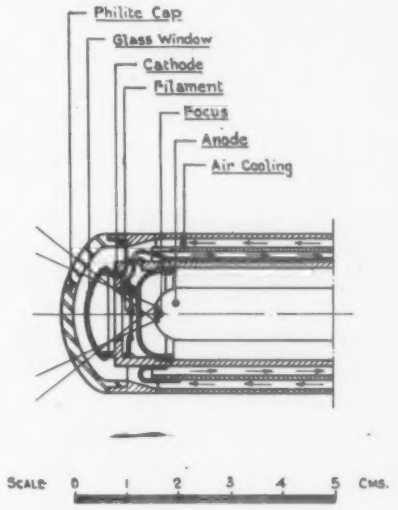


Fig. 4

fined to a small volume of tissue most or all of which is tumor tissue. It was considerations such as these that led Van der Plaats to modify the method of treatment evolved by Chaoul and Adam. By using the Philips tube with its low filtration he was able to confine the energy absorption to a still smaller volume of tissue and so reduce still further the risks of overdosage. The object of his treatment is to give a sufficient dose to the base of the tumor without any consideration of overdosage to the surface but with as little damage as possible to the underlying healthy tissues. For this purpose he uses those factors which provide the steepest decrease of dose with depth obtainable but which allow the application of a sufficient dose at the point intended in a reasonable time. With this method the dose at a depth of 0.5 cm. may be only 40 per cent of the surface dose and the dose at 1.0 cm. depth less than 20 per cent. The

object of treatment is to "burn away" the tumor and yet preserve the surrounding normal tissues and is emphasized by the fact that it is called the "caustic" method of roentgen therapy.

Time Spacing of the Radiation. Following the common practice of high voltage roentgen therapy, Chaoul and Adam adopted fractional irradiation in their treatment. Van der Plaats and others who employ the "caustic" method of roentgen therapy have abandoned fractional irradiation in favor of single massive doses on the grounds that they had difficulty in persuading their patients to attend regularly for treatment to lesions that they considered to be trivial, and further that there was in fact nothing to be gained by fractionation of the dose in this type of treatment. For some very small tumors single doses are effective, but neither the symptom-free rate nor the cosmetic results are as good as those obtained with divided doses and further consideration is necessary before we abandon the established principle of fractionation.

Accessible malignant tumors may be treated either by surgical excision or by destruction *in situ* by irradiation. Success with surgery is dependent upon a sufficiently wide margin of surrounding tissue being removed with the tumor to avoid the possibility of leaving any malignant cells behind. Treatment by radiation relies for its effect upon the malignant cells being more susceptible or less adaptable to this form of injury than normal cells. Excision produces deformity for, by its very purpose, it cannot allow the cosmetic result to be more than a secondary consideration. Deformity following irradiation should be minimal for it is almost entirely due to the previous destruction of the normal tissues by the tumor. Fractionated irradiation is employed to take advantage of the selective action on dividing cells and increase the advantages of irradiation methods over surgical, by minimizing the risk of dissemination and the amount of deformity. As our knowledge of the correct spacing of radiation treatments improves, these advantages will gain even greater importance. Treatment by a single massive dose of radiation sacrifices some of these advantages, it aims at dealing a "vital blow" to the tumor and so falls into an intermediate position between fractionated irradiation and excision. Roentgen therapy aims at destroying the tumor's capacity for proliferation; the dose required is the minimum dose that will prove effective fractionated according to the cell composition and response to treatment of each individual tumor.

Objections to the "Caustic" Method of Roentgen Therapy. The accuracy with which it is necessary to estimate tumor size depends, in practice, on the rate of change of dose with depth in the tissues. It follows that the more we reduce the penetration of our beam the more accurate must our estimation of tumor size become. From tables published by Van der Plaats it may be seen that, under one set of conditions, to deliver a dose of 3,000 r at a depth of 0.5 cm. necessitates a surface dose of 7,500 r, and to deliver the same dose at a depth of 1.0 cm. requires a surface dose of 15,400 r. A difference in depth of 0.5 cm. may therefore mean a change in surface dose of nearly 8,000 r. The intensity of the radiation under these conditions is more than 7,000 r per minute. With such a high intensity and with so rapid a change of dose with depth it is not easy to obtain accurate dosage for the following reasons: (*a*) the very high dosage rate makes the attainment of "saturation" in the ionization chamber difficult, (*b*) the short treatment times necessitate very accurate timing and (*c*) owing to the rapid change of dose with position in the irradiated mass, ionization chambers of normal dimensions will not accurately follow the dosage gradients.

The "caustic" method of roentgen therapy aims at delivering a prescribed dose to the base of the tumor. The base of a tumor is formed by malignant cells spreading out and invading the normal tissues. It is a region in which the percentage of malignant to normal cells is usually decreasing with depth, though the behavior

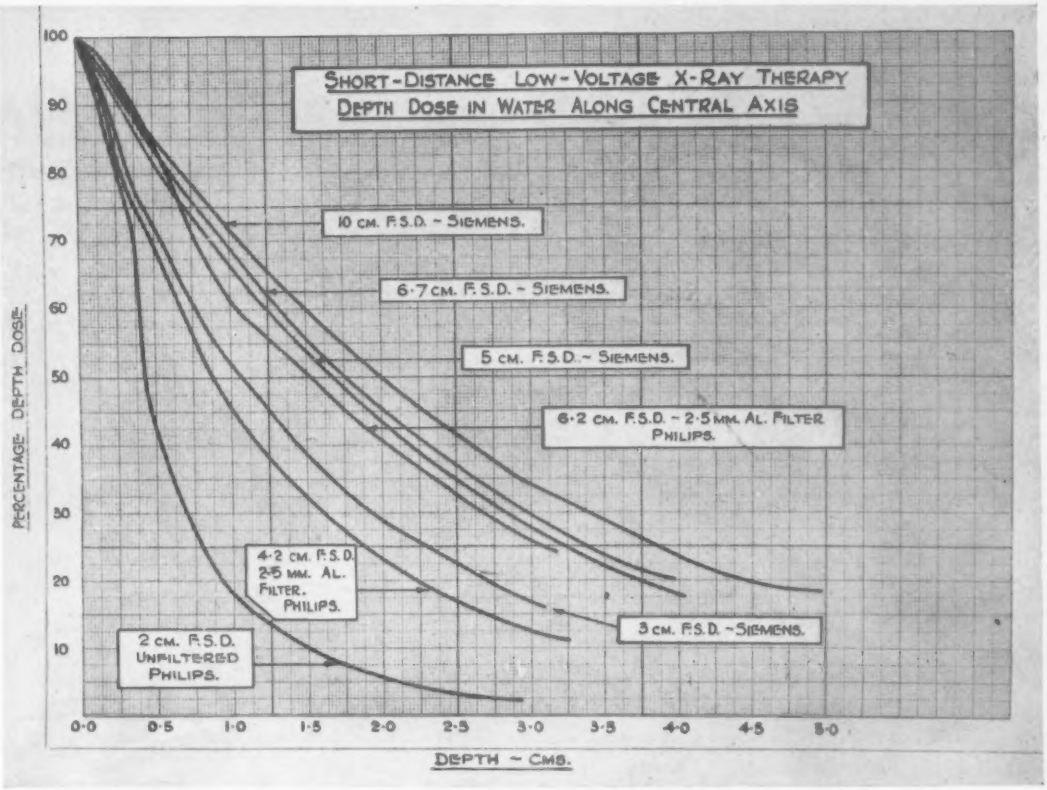


FIG. 5

of the tumor in this region is variable and can only be seen on microscopical examination. For each case treated we must presuppose a change from tumor to normal tissues in the depth commensurate with the rapidity of the change of dose with depth employed. The "caustic" method of roentgen therapy reduces the limits of safety in delivering an adequate dose to the furthermost extension of the tumor. The protagonists of this method have laid great stress on the preservation of the normal tissues from which regeneration must start but healing has been shown to be extremely satisfactory with the methods advocated by Chaoul and Adam so that insistence on still more rapid change of dose with depth in most cases of accessible cancer is unnecessary.

The "caustic" method allows no more than a mere approximation to the dose delivered to the tumor bed. As an irradiation method it sacrifices some of its margin of safety as well as some of its cosmetic advantages to speed and simplicity. It is doubtful whether this is justifiable in the treatment of any but the smallest tumors or except under special circumstances, to be referred to later where speed is of particular importance.

Royal Cancer Hospital Methods. At the

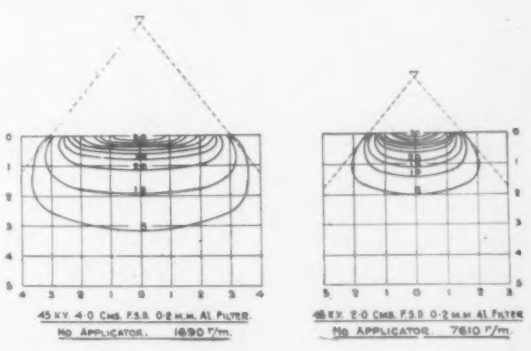


FIG. 6

Royal Cancer Hospital we make use of both the Siemens and the Philips apparatus which together provide a whole series of distributions of the radiation in the short distance low voltage therapy range. These vary from that of the Siemens with a 10 cm. focal-skin distance to the Philips with a 2.0 cm. focal-skin distance (Fig. 5). Treatment is designed on the assumption that the smaller the tumor the less the need for penetration, and the larger the tumor the more the need both for penetration and for fractionation. The most suitable distribution is employed for each particular case.

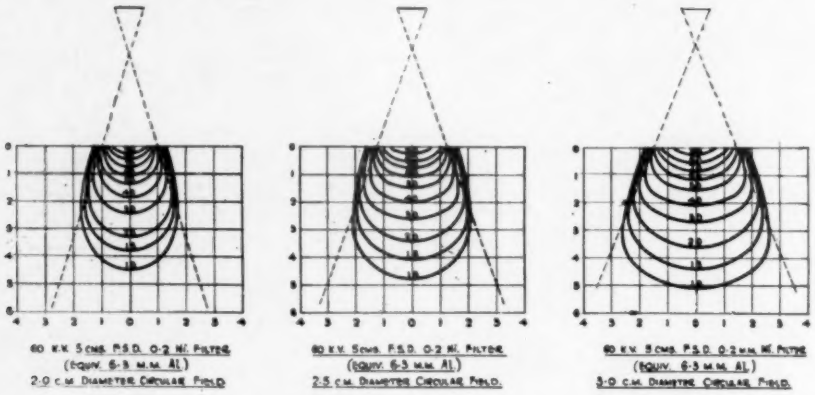

60 K.V. 5 CMS. F.S.D. 0·2 Ni. FILTER.
(EQUIV. 6·3 M.M. Al.)
2·0 C.M. DIAMETER CIRCULAR FIELD.
60 K.V. 5 CMS. F.S.D. 0·2 Ni. FILTER.
(EQUIV. 6·3 M.M. Al.)
2·5 C.M. DIAMETER CIRCULAR FIELD.
60 K.V. 5 CMS. F.S.D. 0·2 M.M. Ni. FILTER.
(EQUIV. 6·3 M.M. Al.)
3·0 C.M. DIAMETER CIRCULAR FIELD.

FIG. 7

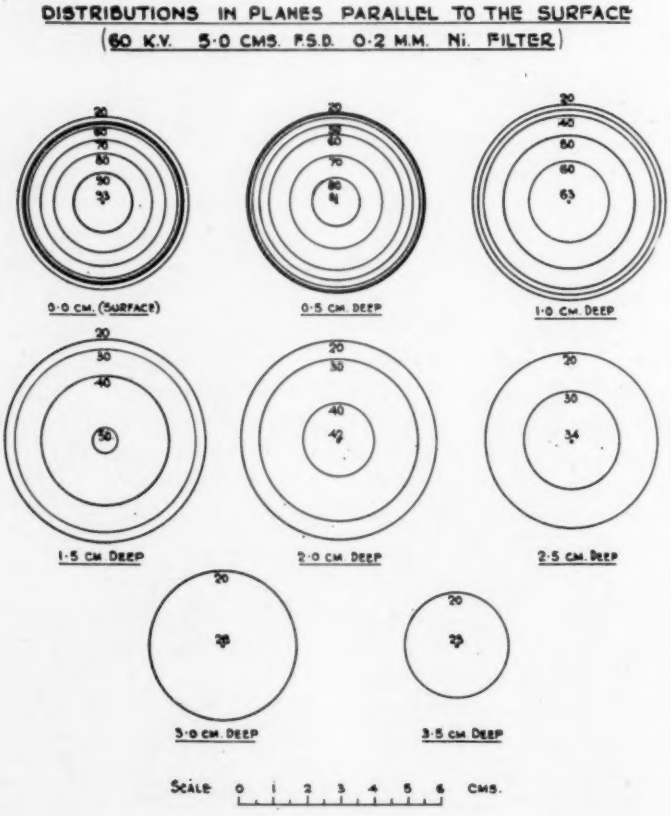

DISTRIBUTIONS IN PLANES PARALLEL TO THE SURFACE
(60 K.V. 5·0 CMS. F.S.D. 0·2 M.M. Ni. FILTER)
0·0 CM. (SURFACE)
0·5 CM. DEEP
1·0 CM. DEEP
1·5 CM. DEEP
2·0 CM. DEEP
2·5 CM. DEEP
3·0 CM. DEEP
3·5 CM. DEEP
SCALE 0 1 2 3 4 5 6 CMS.

FIG. 8

It is the smallest lesions that patients are apt to consider trivial and these may be adequately treated with a single massive dose in some cases. It is not our experience, however, that patients raise any objection to attending for a few minutes each day for treatment, though this is, no doubt due to the fact that the majority of our patients either come short distances or are admitted to the hospital.

In every case a plan of treatment is worked out for each individual patient. The object is to deliver by fractionated irradiation an adequate tumor dose as evenly as possible to a volume that more than includes the limits of the tumor. Whenever necessary, scale drawings are made and isodose curves applied so as to determine the maximum and minimum tumor doses. These isodose curves have been determined by Dr. W. V. Mayneord and Mr. L. F. Lamerton of the Physics Department and are available for all the depth may be estimated from one isodose curve chart (Fig. 9). Larger tumors are covered with cellophane on which their outlines are drawn. An arrangement of

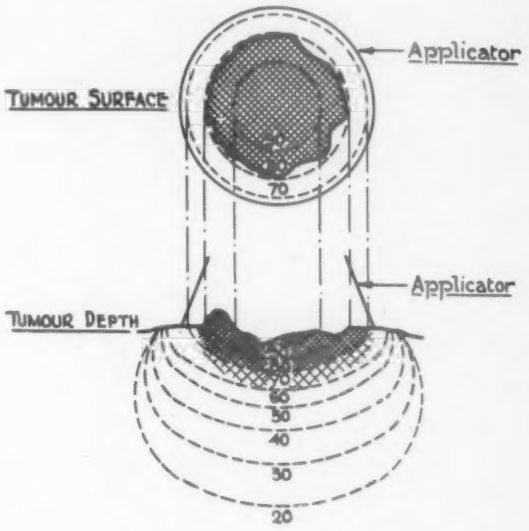


FIG. 9

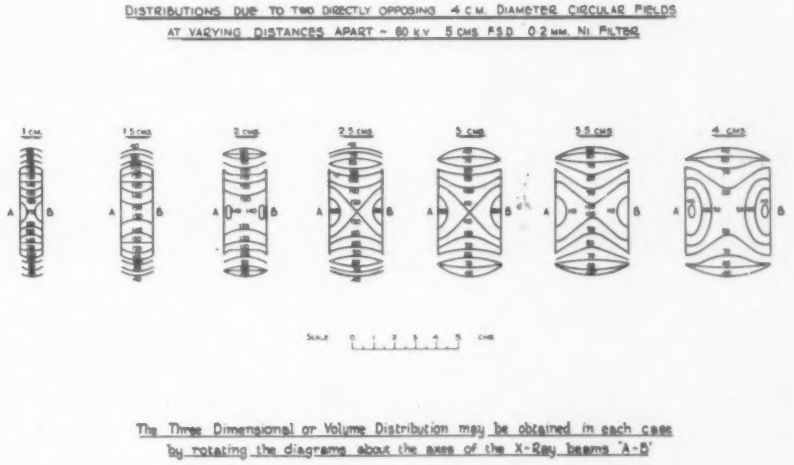
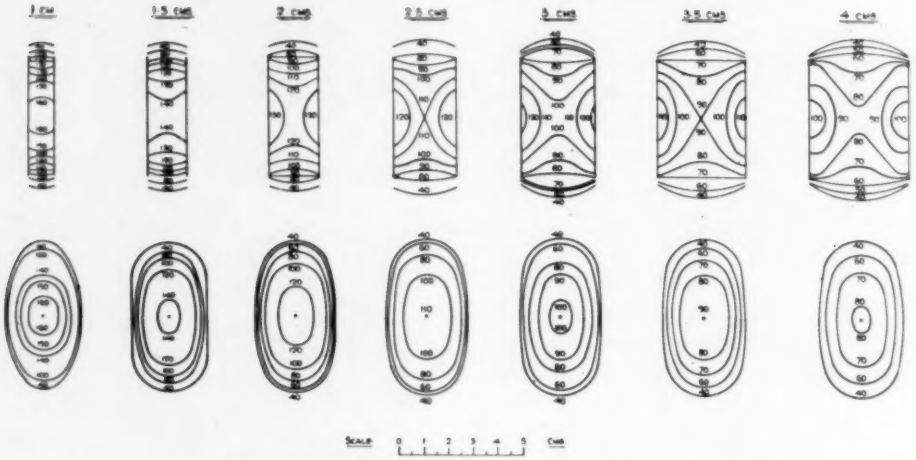

DISTRIBUTIONS DUE TO TWO DIRECTLY OPPOSING 4 C.M. DIAMETER CIRCULAR FIELDS
AT VARYING DISTANCES APART - 60 K.V. 5 CMS. F.S.D. 0.2 MM. Ni FILTER
1 cm.
1.5 cms.
2 cms.
2.5 cms.
3 cms.
3.5 cms.
4 cms.
A
B
SCALE 0 1 2 3 4 5 cms
The Three Dimensional or Volume Distribution may be obtained in each case
by rotating the diagrams about the axes of the X-Ray beams 'A-B'

FIG. 10

conditions used (examples are shown in Fig. 6 and 7). From these a variety of distributions have been drawn and can be referred to at any time. As an example one may instance a series of curves showing distributions at varying short distances below the surface (Fig. 8).

Where a tumor can be covered by a single field the dose on the surface and in the fields that cover the tumor is marked on the cellophane, each field being treated and then covered with a lead disc to prevent re-treatment of overlapping surface areas. By varying the arrangement of the fields daily a fairly even surface dose is delivered. The larger tumors that are accessible but extend to a depth of more than 1 to 1.5 cm. are treated with a roentgen tube operating

DISTRIBUTIONS DUE TO TWO DIRECTLY OPPOSING OVAL FIELDS 4·8 x 2·5 CMS. AT VARYING DISTANCES APART ~ 60 K.V. 5 CMS F.S.D. 0·2 MM. Ni. FILTER.

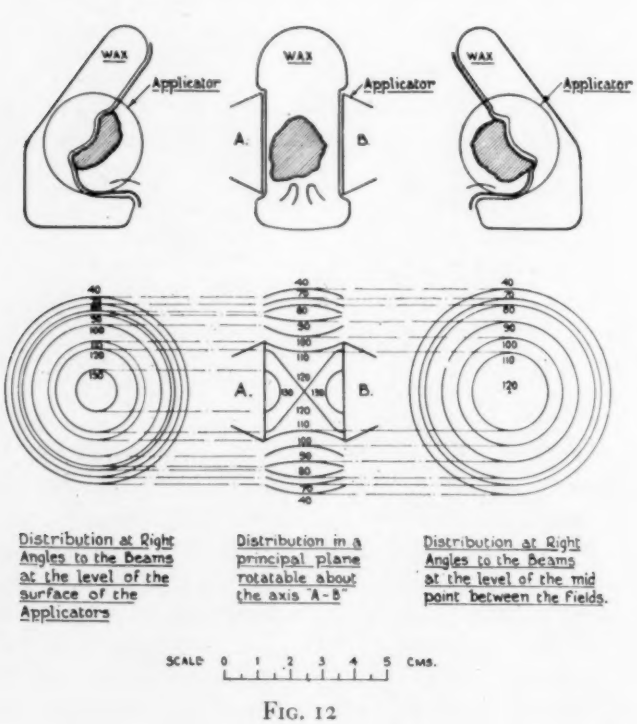


The upper row of diagrams represents the distributions in the principal plane parallel to the beams in the long axes of the applicators. The lower row of diagrams represents the distributions in the plane at right angles to the beams and mid-way between the fields.

FIG. 11

DISTRIBUTION DUE TO TWO 3 C.M. DIAMETER DIRECTLY OPPOSING CIRCULAR FIELDS 2·5 CMS. APART

(60 K.V. 5·0 CMS. F.S.D. 0·2 M.M. Ni. FILTER)



Distribution at Right Angles to the Beams at the level of the surface of the Applicators

Distribution in a principal plane rotatable about the axis A-B

Distribution at Right Angles to the Beams at the level of the mid point between the Fields.

FIG. 12

at 140 kv. with 0.5 mm. Cu filter and a longer focal-skin distance, usually 15 cm. Following the policy of selecting the appropriate distribution for each case a treatment of this kind fills the gap between short distance low voltage and high voltage roentgen therapy. It is in fact middle distance moderate voltage treatment.

Whenever possible, tumors are treated with two directly opposing fields. The distributions due to such fields have been determined for all the varying distances between the fields in common use both for circular (Fig. 10) and for oval fields (Fig. 11). Two opposing fields are used in the treatment of most cases of cancer of the lip, nose, ear, and anterior third of the tongue. In the case of tumors that project irregularly from the surface such as some of those on the nose an impression is taken and a plaster cast made. A strip of wax is moulded on to the plaster cast so that it may fit the patient. With this wax strip in place (Fig. 12), air spaces are eliminated and the tumor is placed in the middle of a homogeneous scattering medium. The position of the fields can be marked on the wax strip and their accurate re-application in the same position assured. In each case the dose given is that which will result in a minimum tumor dose of 4,500–6,500 r for basal and squamous carcinomata in ten to fifteen days.

The average daily dose for single field treatment is 600 r, and a maximum tumor dose of 8,000 r is seldom exceeded.

Eye Protection. Short distance low voltage roentgen therapy is ideally suited for the treatment of tumors in the region of the eyes for the limitation of the irradiation effect itself tends to protect the eyes. When tumors that involve the lids and canthi are to be treated, extra eye protection is necessary. Lead offers the best protection and the voltage employed is not high enough to excite its characteristic radiation. For this purpose we use contact glass or "perspex" lenses to which a 1.0 mm. covering of lead has been applied (Fig. 13). The lead covered lens is inserted under the lids during treatment. These shields transmit less than 0.1 per cent of the radiation; the glass backing offers a non-abrasive surface to the eye, absorbs any β-ray emission

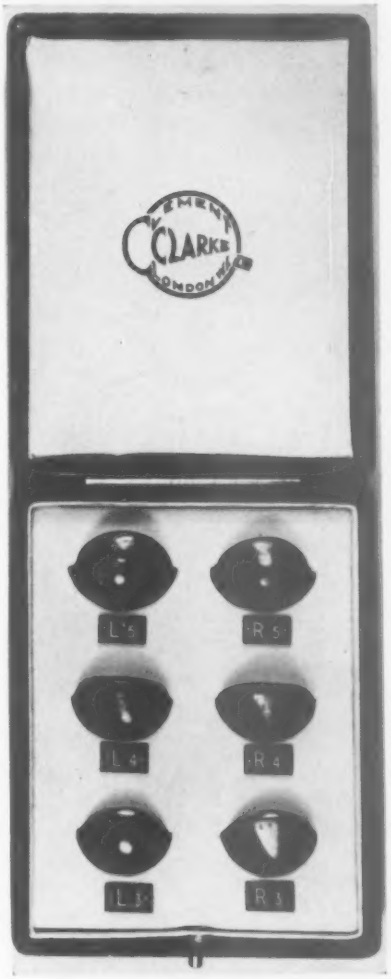


Fig. 13

from the lead and maintains their shape.

Access to Tumor by Surgical Exposure. A special branch of short distance low voltage roentgen therapy is the treatment of tumors made accessible by surgical exposure. It is probable that the scope of this type of treatment will be extended considerably in the future. There are two main methods: (1) the tumor may be exposed long enough for a course of fractionated irradiation, and (2) the tumor may be exposed, a single treatment given and the

wound closed immediately, the patient being anesthetized throughout. The first method has been described by Chaoul with reference to the treatment of carcinoma of the rectum. Dr. P. A. Flood at the Royal Cancer Hospital has treated a number of cases of carcinoma of the bladder by the second method. There are certain obvious advantages about a short exposure where a single treatment can be given under aseptic conditions and it is here that the Philips tube is of special value. Being air cooled it is portable and can be taken to the operating theater, and its great radiation output enables the large single doses necessary to be given very rapidly. Unfortunately, the disadvantages of single massive doses remain.

Intracavitary Therapy. It can be seen from the photographs of the roentgen tubes (Fig. 1 and 3) that they are well designed for insertion into cavities. This type of roentgen therapy was in fact originally designed for the treatment of carcinoma of the cervix. A number of tumors in the mouth can also be treated either by contact therapy or by middle distance moderate voltage treatment with special applicators carrying a side eye piece, light and mirror so that the lesion can be seen during the insertion of the tube and accurate positioning ensured.

Table 1 gives some indication of the scope of the type of treatment described. It represents the work done during the first four years with this treatment at the Royal Cancer Hospital.

TABLE 1

PATIENTS TREATED BY SHORT DISTANCE LOW VOLTAGE ROENTGEN THERAPY FROM JULY 1935 TO JULY 1939

Malignant 466	Non-malignant 294	Total 760

Malignant		*Details of Epitheliomata*	
Epitheliomata	156	Scalp, face and neck	42
Rodent ulcers	134	Lip	31
Carcinomata of breast (mostly skin recurrences)	131	Trunk and limbs	20
Malignant melanomata	8	Buccal mucosa, palate and alveolus	20
Carcinomata of rectum	8	Tongue	17
Carcinomata of bladder	7	Vulva	11
Carcinomata of parotid	3	Ear	10
Carcinoma of lung	1	Penis and scrotum	4
Miscellaneous	18	Peri-anal	1
	466		156

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CONCENTRATION METHOD OF RADIOTHERAPY FOR CANCER OF THE MOUTH, PHARYNX AND LARYNX*

REPORT OF PROGRESS

By MAX CUTLER, M.D.
CHICAGO, ILLINOIS

THIS report of progress presents the results achieved so far by the new method of irradiation called concentration radiotherapy.[1] During the five year period, April, 1938, to April, 1943, 290 cases have been treated, divided as follows:

Cancer of the pharynx and larynx	116 cases
Cancer of the mouth	116 cases
Cancer of the tongue	38 cases
Cancer of the accessory nasal sinuses	20 cases
Total	290 cases

As originally stated, the purpose of these studies is to improve the effectiveness of external irradiation in the more radioresistant types of cancer of the mouth, pharynx, and larynx by systematic controlled clinical research in the basic factors of radiotherapy. Specifically, the following questions are of special interest:

(1) The comparative value of roentgen rays and radium.
(2) The optimum voltage.
(3) The optimum roentgen intensity.
(4) The optimum daily dose, total dose, and treatment time.
(5) The optimum size and number of fields.

The only manner in which these basic questions can be answered is to treat comparable groups of cases under controlled conditions by techniques in which only one factor is varied at a time. This is an immense task and has hitherto not been attempted. The staff of the Chicago Tumor Institute first undertook to study these problems in April, 1938.

After testing numerous variations in the technique of external irradiation, several methods have crystallized which seem to be the most effective. Each technique is applicable to a certain type of lesion as regards location and extent. These techniques are in no way considered as final, but they constitute a definite improvement in the control of the more radioresistant cancers over the former methods. For the purpose of research, it was decided to adopt these several methods and apply them to comparable groups of cancer of the mouth, pharynx, and larynx, varying only one factor at a time. In this manner, it is hoped to evaluate the various basic factors of radiotherapy. For example, three groups of intrinsic carcinoma of the larynx are now treated with roentgen rays and telecurietherapy in which all the factors are the same except that one group is treated with 400 kv. roentgen rays, another with 200 kv. roentgen rays, and a third with telecurietherapy, with the view of ultimately learning the comparative value of the three methods. In a similar manner the factors of roentgen intensity, daily dose, total dose, and total treatment time are being studied.

The following five techniques have been adopted in this research:

(1) *Telecurietherapy, Twelve Treatment Days, Single Constant Portal, Dose 120,000 mg-hr.* This technique is used in lesions of the alveolar ridge and floor of the mouth in which the disease is entirely or mainly unilateral. The following is an example:

CASE 1. Male, aged seventy-three, ulcerated lesion of mucous membrane right upper alveolar ridge extending on to the right cheek. No adenopathy. Biopsy—squamous carcinoma. Treatment—telecurietherapy (10 grams of radium), single right lateral field, 6 by 6 cm., distance 12.5 cm., filtration 1 mm. platinum,

* From the Chicago Tumor Institute and the Hines Veterans Hospital aided by a Grant from the National Cancer Institute.

5,000 mg-hr. twice daily for twelve days, total 120,000 mg-hr. between April 7, 1941, and April 19, 1941. At the height of the reaction there developed a pronounced "epithelitis" of the mucous membrane and a mild epidermitis. On June 5, 1941, the lesion had disappeared and there is no evidence of disease at this time, two years and four months after completion of treatment.

Comment. The interesting feature in this case is the use of a large dose over a comparatively short period (twelve days) through a single portal. A similar group of lesions is being treated with roentgen rays in order to compare the results.

FIG. 1. Case III. Epidermitis first appeared on the twenty-second day from the beginning of treatment and healed ten days later.

(2) *Roentgen Therapy, Twelve Treatment Days, Two Constant Portals, Dose 8,400 r.** This technique is used in lesions of the soft and hard palate. The following is an example:

CASE II. Male, aged fifty-two, ulcerated lesion of left soft palate measuring about 5 by 5 cm. No adenopathy. Biopsy—epidermoid carcinoma transitional cell type.† Treatment—July 7, 1941 to July 19, 1941, 400 kv. roentgen rays, 5 ma., 5 mm. copper, 85 cm. distance, 6 by 6 cm. portals, 5.2 to 6.8 r per minute, 350 r to each side daily and consecutively for twelve treatment days, 4,200 r to each lateral portal, total dose 8,400 r, measured on the skin. Marked "epithelitis" of the mucous membrane but no epidermitis. The lesion disappeared one month after completion of treatment. There is no evidence of recurrence at this time, two years later.

Comment. The interesting feature in this case is the ability to give a very large dose (8,400 r) in a short period of twelve treatment days without untoward local reactions and with restoration of apparently normal conditions in the skin and in the tissues of the tumor bed. The complete disappearance of the lesion in this case is at least partly due to its radiosensitivity since the microscopic structure showed transitional cell carcinoma, but similar results have been observed with adult squamous carcinoma of the palate.

(3) *Roentgen Therapy, Eleven Treatment Days, Single Diminishing Portal, Dose 5,400 r.*‡ This technique is used in intrinsic carcinoma of the larynx affecting the true or false cord or both structures in which for various reasons it is decided to use radiotherapy. The following is an example:

CASE III. Male, aged thirty-eight, ulcerated lesion occupying the left true cord, touching the anterior commissure and almost reaching the posterior commissure. The lesion was rather bulky and extended into the lumen of the larynx, on to the left false cord and subglottically. The left hemilarynx was almost completely fixed. The left arytenoid was partly fixed. The right hemilarynx was normal. Biopsy—squamous carcinoma. Treatment—June 14, 1942 to June 26, 1942, 400 kv. roentgen rays, 5 ma., 5 mm. copper, distance 84 to 115 cm., single left lateral portal 5 by 5 cm. gradually diminished to 3 by 3 cm. Roentgen intensity 4 r per minute to 3.3 r per minute. Two equal treatments daily beginning with 100 r twice daily and increasing to 425 r twice daily, total dose 5,400 r, measured on the skin. After a twelve day interval, the following supple-

* This dose is too strong for some patients and has been reduced to 7,600 r.

† Because of the transitional cell type of lesion, the result in this case cannot be attributed exclusively to the type of irradiation as the lesion may have responded to less intensive treatment.

‡ In some instances an additional 2,000 r has been given to the opposite side about two weeks after the end of the series, as in the case described.

mentary treatment was given to the opposite side of the larynx: 500 r twice daily for two days through a 3 by 3 cm. portal, dose 2,000 r, grand total 7,400 r. The patient developed an "epithelitis" and epidermitis (Fig. 1). The lesion disappeared slowly, no longer being visible five weeks after the end of the main cycle of irradiation. There is no evidence at this time, two years after completion of treatment.

Comment. There are several interesting features about this case. The lesion was operable and probably curable by laryngectomy. It was too advanced for laryngofissure. The patient is a teacher and public speaker, hence he asked for a chance of cure without laryngectomy. The fact that the left hemilarynx was still slightly movable finally led to the decision to try irradiation as the initial procedure. The interesting features of the technique are the use of a single portal, avoidance of using portals that are unnecessarily large, the gradual diminution of the portal with corresponding increase in the daily dose, and the comparatively large total dose in a relatively short treatment period. The appearance of the larynx five weeks after treatment was entirely normal, the mobility of the cords was fully re-established, the voice was normal, and the patient regained the 25 pounds he had lost. His morale was exceptionally high as he had been told that cure without laryngectomy was not possible.

(4) *Roentgen Therapy, Interrupted Method, Ten Treatment Days, Two Fields, Diminishing Portals, Dose 7,700 r.** This technique is used in more advanced intrinsic carcinoma of the larynx in which the advantages of the therapeutic test are desired. In certain operable intrinsic lesions this method of interrupted treatment permits one to estimate the probable radiosensitivity of the lesion before the second phase of the irradiation is given. In this manner, the second cycle can be omitted if the lesion appears to be relatively radioresistant and laryngectomy can be resorted to. The following is an example:

* A third cycle consisting of 2,000 r in two days is sometimes added as in the case reported here.

Case iv. Male, aged forty-three, continuous and increasing hoarseness for one year, pain on swallowing for three months. Examination—right true and false cords are the seat of a large ulcerated mass projecting into the lumen of the larynx with almost complete fixation of the right hemilarynx (Fig. 2). Biopsy—squamous carcinoma. For six days (February 24, 1941 to March 1, 1941) the patient received two treatments daily as follows: 400 kv. roentgen rays, 5 mm. copper filter, 85 cm. distance, 5 ma., portals 30 to 20 sq. cm. Doses: first day 400 r, second day 500 r, third day 550 r, fourth day 650 r, fifth day 700 r, sixth day 800 r, total dose 3,600 r, measured on the skin. Roentgen intensity varied between 6.3 r per minute and 8.9 r per minute. The first cycle of treatment

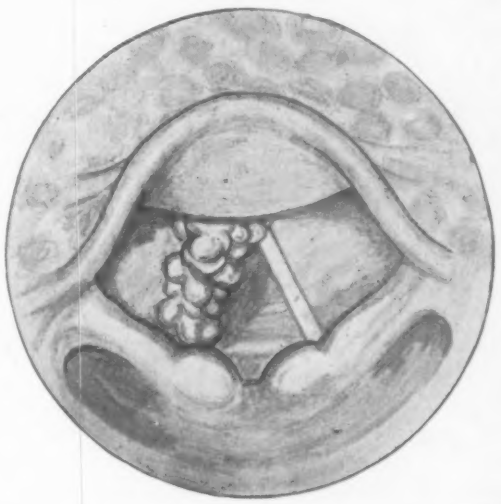

Fig. 2. Case iv. Squamous carcinoma of the right true and false cords. (Drawing by D. Holvey, M.D.)

was given over the right side of the larynx. On March 12, 1941, eleven days after completion of the first roentgen-ray cycle, the lesion showed the first sign of regression and there was more mobility of the right hemilarynx. Two days later, the thirteenth day after the last treatment, there was marked further regression and increased mobility.

Because of this pronounced improvement, it was decided to administer the second cycle of roentgen-ray treatments. The interval between the two cycles was twelve days. The second cycle began March 14, 1941, and finished March 17, 1941, with the following factors: 400 kv. roentgen rays, 5 mm. copper, 85 cm. distance, 5 ma., portals 30 sq. cm. to 12 sq. cm.

Doses: first day 850 r, second day 950 r, third day, 1,050 r, fourth day, 1,150 r, two treatments daily, total dose 4,000 r, measured on the skin. The total dose during the second cycle was given over the left side of the larynx. The first cycle was 3,600 r and the second cycle 4,000 r, making a total of 7,600 r for the two cycles during ten treatment days.

The maximum reaction on the skin consisted of a deep pigmentation. The maximum mucous membrane reaction was a very mild "epithelitis." The mobility of the right hemilarynx was restored to normal on April 4, 1941, eighteen days after the completion of the second cycle of irradiation. The lesion continued to regress and finally disappeared on April 25, 1941, thirty-eight days after the last treatment of the second cycle, but there remained a small, irregular, non-ulcerated nodule about 6 mm. in diameter situated on the posterior extremity of the right true cord. On May 9, 1941, fifty-three days after the last treatment, it was decided to administer a third course of irradiation. It was not possible to decide clinically whether or not this nodule contained a remnant of carcinoma and it was felt that a biopsy should not be done. From previous experience, it seemed that a third course of irradiation could be given with safety. Consequently, the third course was administered in two days as follows: Field over right side of larynx, 400 kv. roentgen rays, 5 mm. copper, 80 cm. distance, 7 sq. cm. portal, 7.4 r to 9.8 r per minute, 500 r twice daily for two days or a total of 2,000 r, measured on the skin. The grand total for the three cycles of irradiation was 9,600 r. The last treatment was given on May 10, 1941. The nodule regressed slowly and finally resolved into a small fibrotic thickening about 3 mm. in diameter which has remained stationary for three years. It is obvious that this is an area of scar tissue. On April 29, 1942, the patient had gained about 20 pounds in weight, the voice was normal, and the right hemilarynx was normal in appearance and mobility.

Comment. There are several significant features about this case. The most important is the disappearance of an extensive intrinsic squamous carcinoma of the larynx with almost complete fixation of the hemilarynx, with freedom from recurrence for three years. Several features of the treatment are interesting. The large total dose (9,600 r) administered in only twelve treatment days was possible with very little reaction in the skin and mucous membrane. The division of the treatment into three cycles with two intervals afforded an opportunity to observe the response of the lesion and plan further treatment accordingly. Finally, this case illustrates that it is practical to test the radiosensitivity of a lesion with a partial irradiation and use such information as a guide to further treatment.

For some patients this treatment is too intense and has resulted in radionecrosis, hence the doses have been reduced about 20 per cent.

(5) *Roentgen Therapy, Eighteen Treatment Days, Single Diminishing Portal, Dose 6,500 r.* This technique is used in extrinsic carcinomas of the larynx (pyriform fossa, aryepiglottic fold, epiglottis). The following is an example:

CASE V. Male, aged fifty-seven. There is an extensive swelling over the region of the thyroid and cricoid obliterating the normal contour of the anterior surface of the neck (Fig. 3*A*). There is a marked swelling and ulceration in the region of the right true and false cords, extending to the subglottic region. The right arytenoid is enlarged, the right hemilarynx is partly fixed. There is no cervical adenopathy. Biopsy showed squamous carcinoma. Soft tissue roentgenography of the larynx disclosed extensive destruction of the cricoid and thyroid cartilages. Treatment was given between July 7 and July 30, 1938, over a period of twenty-one treatment days, one treatment daily through a single right lateral field, 400 kv., 5 ma., 8 mm. copper, distance varied between 65 cm. and 90 cm., size of portal varied between 8 by 10 cm. and 3 by 4 cm., daily dose increased gradually from 100 r to 700 r, measured on the skin, roentgen intensity varied between 4.1 r per minute and 7.5 r per minute. Total dose 6,500 r. The tumor regressed rapidly during the first few days of treatment. Approximately one week after beginning treatment, an abscess pointed over the thyroid region which was incised and evacuated. This resulted in marked relief. The weight increased and the general condition improved. On October 6, 1938, about ten weeks after the comple-

tion of treatment, there was no definite evidence of disease, the general condition was excellent, and the patient had no complaints. Subsequent examination of soft tissue roentgenograms of the larynx disclosed a remarkable restoration of the cartilages to what seems to be a normal state. There is no sign of recurrence at this time, about six years after completion of treatment (Fig. 3*B*).

Comment. This technique* is the most effective so far observed for the more extensive intrinsic carcinomas and for extrinsic carcinomas of the larynx. When the cervical adenopathy has been too extensive to be included in the fields, an additional cycle of treatment has been given to such areas as could not be included in the first cycle of treatment.

A *B*

Fig. 3. Case v. *A*, extensive carcinoma of the right hemilarynx with perforation through the thyroid and cricoid cartilages and obliteration of the normal contour of the neck before treatment. *B*, five years after treatment showing restoration of the normal contour of the neck.

DISCUSSION

The basis of the method of concentration is the use of large daily doses over a comparatively short treatment period (ten to eighteen days). The total dose is sufficient to produce an "epithelitis" and in some instances an epidermitis and there is reason for believing that the total dose under the given conditions constitutes a maximum irradiation. It is by no means certain that all the factors are the most favorable, except that *the increase in the daily dose and diminution in the total treatment period has caused regression, disappearance, and apparent cures of lesions that have failed to respond to the former divided dose technique.* There is reason to expect that the technique of gradually increasing the daily dose and diminishing the size of the field is of advantage but this principle cannot be adequately supported on theoretical grounds and the data at hand are not sufficient to settle the question definitely at this time. The idea of delivering the largest doses through the smallest fields in the last few days is intriguing, and for certain types of carcinomas, as in the larynx, this technique appears to be especially effective.

Before 1922 surgery was the only form of treatment for operable carcinomas of the larynx. The first successful results of radiotherapy were reported by Regaud, Coutard and Hautant[5] who presented 6 inoperable cases treated successfully with roentgen therapy. In 1938 Jackson and Jackson[2] stated their belief that the future will prob-

* This was the first case treated by this method and the treatment time was twenty-three days. The treatment time has now been reduced to eighteen days and two treatments are used daily instead of one. The total dose remains the same.

ably see a progressive decrease in the relative number of laryngectomies and an increase in the number of patients treated with irradiation. Similar views have been expressed by Quick[4] and others. Lenz[3] has published favorable results of roentgen therapy in cancer of the larynx.

As long as radiotherapy was limited to inoperable lesions, the problem of choosing between operation and irradiation was relatively simple. Now that irradiation is considered to be the method of choice in certain operable lesions, the problem has become much more difficult. The responsibility of advising radiotherapy instead of laryngectomy in certain conditions in which the opposing factors appear to be equally balanced is indeed a serious matter. The knowledge that if radiotherapy fails, laryngectomy may still be performed with safety is of considerable help. As experience accumulates, the decision becomes less difficult. Intrinsic lesions of the true vocal cord

TABLE I

CARCINOMA OF LARYNX
(True Cord)

Early—cord movable	35%*
Border line—cord partly movable	35%
Advanced—cord fixed	30%

Year	Cases Treated	Alive at End of				
		1939	1940	1941	1942	May 1943
1938	7	5	5	5	5	4
1939	7	7	5	5	5	5†
1940	9		7	6	5	5
1941	21			21	17	15
1942	12				12	10‡
1943	1					1
	57					40

* These figures represent the percentages of the total number of cases included in the table.
† Laryngectomy followed radiotherapy in 1 case.
‡ Cardiac death, no evidence of cancer in 1 case; laryngectomy followed radiotherapy in 2 cases.

TABLE Ia

CARCINOMA OF LARYNX
(True Cord—Movable and Partly Fixed)

Year	Cases Treated	Alive at End of				
		1939	1940	1941	1942	May 1943
1938	7	6	5	5	4	4
1939	4	4	3	3	3	3
1940	6		5	5	4	4
1941	13			13	11	11
1042	9				9	9
1943	2					2
	41					33

TABLE Ib

CARCINOMA OF LARYNX
(True Cord, Movable)

Year	Cases Treated	Living and Well	Died in		
			1939	1940	1942
1938	2		1		1*
1939	3	2		1	
1940	3	3			
1941	7	7			
1942	3	3			
1943	2	2			
	20	17			

Seventeen living and well, average length of time since treatment—25 months.

* Died of cerebral metastasis—primary lesion controlled.

TABLE II

CARCINOMA OF LARYNX AND HYPOPHARYNX
(Epiglottis, False Cord, Pyriform Fossa)

Year	Treated	Alive at End of				
		1939	1940	1941	1942	May 1943
1938	11	5	3	3	2	2
1939	6	6	4	3	3	3
1940	4		3	2	2	2
1941	18			18	14	10*
1942	18				18	17
1943	1					1
	58					35

Sixty-two per cent of lesions were advanced and associated with cervical adenopathy.

* Laryngectomy following radiotherapy in 1 case.

with subglottic extension and complete fixation have failed to respond to the most intensive radiotherapy; consequently laryngectomy is performed in these cases if there are no contraindications. When the fixation of the vocal cord is only partial, irradiation is usually the initial procedure.

TABLE III

CARCINOMA OF MOUTH, OROPHARYNX AND NASOPHARYNX

(Excluding Tongue)

Early	10.5%
Advanced	34.5%
Very advanced	55%

Year	Cases Treated	Alive at End of				
		1939	1940	1941	1942	May 1943
1938	13	6	5	5	3	3
1939	15	9	5	5	4	2
1940	17		15	11	8	8
1941	35			28	22	21*
1942	27				23	21
1943	9					9
	116					64

* Excision of remnant in 1 case; neck dissection after radiotherapy in 1 case. Five cases died of causes other than cancer (clinically free of disease).

TABLE IV

CARCINOMA OF TONGUE

Early	13%
Advanced	39%
Very advanced (with adenopathy)	48%

Year	Cases Treated	Alive at End of				
		1939	1940	1941	1942	May 1943
1938	5	0	0	0	0	0
1939	2	2	0	0	0	0
1940	8		6	4	2	2
1941	16			14	10	10*
1942	6				5	4†
1943	1					1
	38					17

* Hemiglossectomy following radiotherapy in 1 case.
† Neck dissection and hemiglossectomy following radiotherapy in 1 case.

TABLE V

CARCINOMA OF ACCESSORY NASAL SINUSES

Year	Cases Treated	Alive at End of				
		1939	1940	1941	1942	May 1943
1938	6	4	4	4	3	3
1939	2	1	1	1	1	1
1940	0					
1941	8			5	3	3
1942	3				3	3
1943	1					1
	20					11

The accompanying tables give the results of treatment of the 290 cases which form the subject of this research.

SUMMARY

(1) A method of radiotherapy has been evolved which is called the concentration method. Roentgen rays or radium may be used. This method has resulted in the eradication of the more radioresistant carcinomas of the mouth, pharynx, and larynx which have failed to respond to the previous methods of external irradiation.

(2) As a result of this development, the initial disappearance and apparent cures have been observed in a group of intrinsic squamous carcinomas of the larynx which are generally regarded as radioresistant and for which surgery is usually claimed to be the only effective method of treatment. In many of these cases the only alternative to radiotherapy was complete laryngectomy.

(3) Although it is necessary to treat more cases and wait still longer in order to be more certain of the permanency of the results, it is already clear that the surgical attitude toward the treatment of some forms of intrinsic cancer of the larynx must, on the basis of these results, be altered.

(4) A radiotherapeutic test has been developed for certain border line cases of intrinsic cancer of the larynx in which a decision between radiotherapy and laryngectomy is difficult. Approximately half the

full dose of treatment is given in six days as a test of the sensitivity of the lesion. This is followed by an interval of fifteen days of observation. The degree of regression of the lesion at the end of the twenty-one days can be used as a reasonably accurate index of the radiocurability of the lesion. If the response to the first course of treatment is adequate, the second course is administered. If the response has been inadequate, laryngectomy may be performed with safety. This method has been a great help in certain difficult border line cases and gives the patient a chance of cure with preservation of his voice in the event the lesion proves to be sensitive to irradiation. It also does not eliminate his chances of cure by laryngectomy if the lesion proves to be radioresistant.

(5) Five different techniques of irradiation have been developed under the method of concentration. Each is applicable to a certain type of cancer of the mouth, pharynx, and larynx.

(6) The results in radioresistant forms of cancer of the mouth have been improved by the method of concentration.

(7) A study of the comparative value of 220 kv. roentgen rays, 400 kv. roentgen rays, and radium has been started. The study has not proceeded far enough to justify an opinion at this time.

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Vol. 51, No. 6

RADIUM DOSAGE FOR LINEAR SOURCES

TABLE AND NOMOGRAM*

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IN a previous article,[3] data on dosage for linear sources of radium, calculated by the method of Laurence,[1,2] were presented in graphical form. Because of difficulties in graph reading, the data are here presented in two simpler forms—a table and a nomogram.

Table 1 gives directly the dose in gamma roentgens (r_γ) per milligram-hour along the "end" axis, "in air," for filtrations equivalent to 0.5 mm. platinum. Other filtrations and tissue absorption are considered later. By "end" axis is meant the line perpendicular to the linear source at its end, e.g., *BN* in Figure 1 and not the line which would represent the prolongation of the linear source. In reality, the "end" axis is a plane, but appears in the diagram as a line. "Distance" refers to the distance measured along this or other perpendicular axes in centimeters. The distribution of the radium salt in the linear source is assumed to be uniform.

The value 9.0 was used as the number of gamma roentgens from a 1 milligram point source of unfiltered radium element at 1 centimeter in 1 hour.

A dosage table for points along an end axis is valuable because it can be used to obtain the dose at all points. This arises from the fact that a perpendicular line from any given point to a linear source may be considered an end axis for the two linear segmental sources into which the original or true linear source is divided by this line. The following examples will make this clearer and illustrate the use of the method. An active length of 4 cm. and a treatment distance of 2 cm. are arbitrary figures selected only for convenience of demonstration.

Assume a linear source of active length 4 cm. (*AB* of Fig. 1, 2 and 3) and filtration equivalent to 0.5 mm. Pt:

Example 1. Find the dose per milligram-hour 2 cm. away from the linear source along the end axis, i.e., at point *N* (Fig. 1).

The dose at point *N* is read directly from Table 1 as 1.10 r_γ per milligram-hour by following the vertical line under active length 4 and a horizontal line from distance 2.

Example 2. Find the dose per milligram-hour 2 cm. away from the linear source along the "central" axis, i.e., at *M* (Fig. 1). A central axis is a line perpendicular to the linear source at its center.

The line *OM* may be assumed to be the

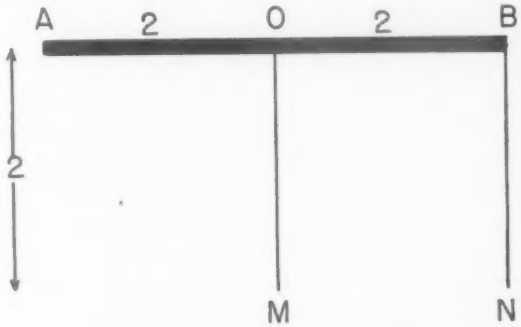

FIG. 1. *AB* is the active length of the linear source; *BN* is the end axis; *OM* is the central axis. The dose at point *N* is calculated in example 1. The dose at point *M* is calculated in example 2.

end axis for the two segmental linear sources *AO* and *BO*, each of which contributes 0.5 milligram-hour if the total contribution from *AB* is 1 milligram-hour.

The dose at *M* from the linear segment $AO = 1.62/2 = 0.81$ r_γ for 0.5 milligram-hour (1.62 is the dose per milligram-hour from the linear source *AO* obtained from Table 1. This figure must be divided by 2 since *AO* contributes only one-half of one milligram-hour).

The dose at *M* from *BO* is the same as the dose from *AO*. Therefore the dose at *N* from both *AO* and *BO*, i.e. from *AB*, equals

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twice the dose due to *AO* alone for twice the number of milligram-hours from *AO* alone, or:

Dose due to $AB = 2 \times 0.81$ or $2 \times 1.62/2 = 1.62$ r_γ for 1 mg-hr.

It turns out that we have both divided and multiplied the dose from *AO* by 2 or that the dose per milligram-hour along the central axis of a linear source 4 cm. long is equal to the dose per milligram-hour

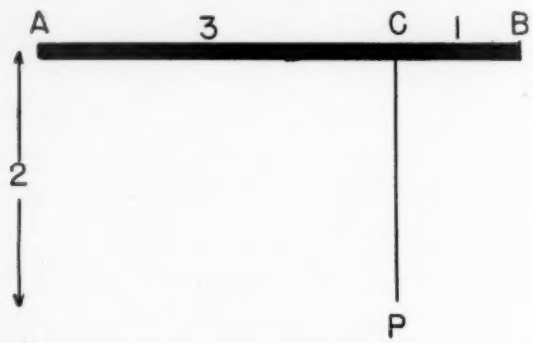

FIG. 2. *AB* is the active length of the linear source; *CP* is the perpendicular axis through a point 1 cm. from the end *B* and 3 cm. from the end *A*. The dose at point *P* is calculated in example 3.

along the end axis of a source 2 cm. long at the same treatment distance. This is the general rule for points on a central axis. Therefore, one can find the dose per milligram-hour at any point along a central axis by looking up in the table under one-half the active length instead of the full active length. Calculations for this type of case are therefore unnecessary and the table is used directly.

Example 3. Find the dose per milligram-hour 2 cm. away from the linear source along an axis 3 cm. from one end and 1 cm. from the other end, i.e., at *P* (Fig. 2). The two segmental linear sources are *AC* and *BC* (Fig. 2).

For purposes of calculation in cases of this type, it is usually simpler to assume that the number of milligram-hours represented by the linear source itself and each of its segments is equal to the number of centimeters in its length instead of assuming that the linear source represents 1 mg-hr. and each segment a fraction thereof depending on its length. This assumption is possible because the linear density of the radium salt in the capsule or needle is uniform and therefore the number of milligram-hours contributed by each segment is proportional to its length.

Assume that the segment *AC*, being 3 cm. long, contributes 3 mg-hr. The segment *BC*, 1 cm. long, contributes 1 mg-hr.

From the table, we find that the dose per milligram-hour from the segment *AC* (active length 3 cm.) at the point *P* (distance 2 cm.) is 1.34 r_γ. For 3 mg-hr., this figure must be multiplied by 3:

Dose at *P* from *AC* for 3 mg-hr. $= 1.34 \times 3 = 4.02$ r_γ.

Similarly, the dose at *P* from *BC* for 1 mg-hr. $= 1.88 \times 1 = 1.88$ r_γ.

When we add the dose from *AC* (for 3 mg-hr. of *AC*) to the dose from *BC* (for 1 mg-hr. of *BC*), we obtain the dose from

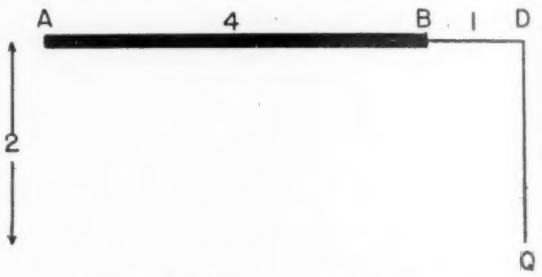

FIG. 3. *AB* is the active length of the linear source; *DQ* is the perpendicular axis through a point 1 cm. beyond the end *B*. The dose at point *Q* is calculated in example 4.

AB, i.e., from the true linear source (for 4 mg-hr. of *AB*):

Dose at *P* from *AB* for 4 mg-hr. $= 4.02 + 1.88 = 5.90$ r_γ. The figure 4 mg-hr. is of course the sum of 3 and 1 mg-hr. and is also equal to the total active length in centimeters.

Since we know the dose for 4 mg-hr., we can easily find the dose for any other number of milligram-hours. For 1 mg-hr., the dose $= 5.90/4 = 1.48$ r_γ.

Example 4. Find the dose per milligram-hour 2 cm. away from the linear source along an axis 1 cm. beyond the end of the linear source, i.e., at *Q* (Fig. 3). In this

case, the two "segments" are *AD*, 5 cm. long, and *BD*, 1 cm. long. (The "segment" *AD* is actually larger than the linear source itself, but the term segment is still used by analogy with the previous case.) We assume that *AD* contributes 5 mg-hr. and *BD* 1 mg-hr. Clearly, in this example, the dose from *BD* must be subtracted from the dose due to *AD* in order to obtain the dose from *AB*, the true linear source.

Dose at $\mathcal{Q}$ from *AD* for 5 mg-hr. $=.94\times5=4.70$ r_γ.

Dose at $\mathcal{Q}$ from *BD* for 1 mg-hr. $=1.88\times1=1.88$ r_γ.

Dose at $\mathcal{Q}$ from *AB* for 4 mg-hr. $=4.70-1.88=2.82$ r_γ. The figure 4 is the difference between 5 and 1 mg-hr. and is equal to the number of centimeters in the total or true active length.

Dose at $\mathcal{Q}$ per milligram-hour of *AB* $=2.82/4=0.71$ r_γ.

The data included in the table may also be put in the form of a nomogram. This is an alignment chart (Fig. 4) consisting of two vertical scales and a central oblique scale. The vertical scale on the left is called the *C* scale; the vertical scale on the right, the *D* scale, and the central oblique scale, the *F* scale. The advantages of a nomogram are that it is compact and that all intermediate values are easily obtained. Any linear source is looked upon as consisting of two separate linear sources or segments, as described in the previous examples. Each of these two segments is treated separately in succession by the nomogram in exactly the same way. On the left vertical scale (*C* scale), one finds the active length of the

Fig. 4. Nomogram to calculate dosage for linear sources. For explanation, see text.

segment. On the right vertical scale (*D* scale), one finds the treatment distance. Then, a straight edge—e.g., a ruler, preferably transparent, or a piece of string stretched tightly—is placed across the chart through the two selected points on the *C* and *D* scales. This straight edge intersects the central oblique scale at a point which gives an *F* figure for each of the two segments. The two *F* figures thus obtained are then added—or subtracted if the point in question lies beyond the end of the linear source (as in *Example 4* above). One further step is necessary when the nomogram is used. Because of the form of the fundamental equation for dosage from linear sources, the result of the addition or subtraction of the two *F* figures must be divided by the total active length and by the treatment distance before the dose per milligram-hour is obtained.

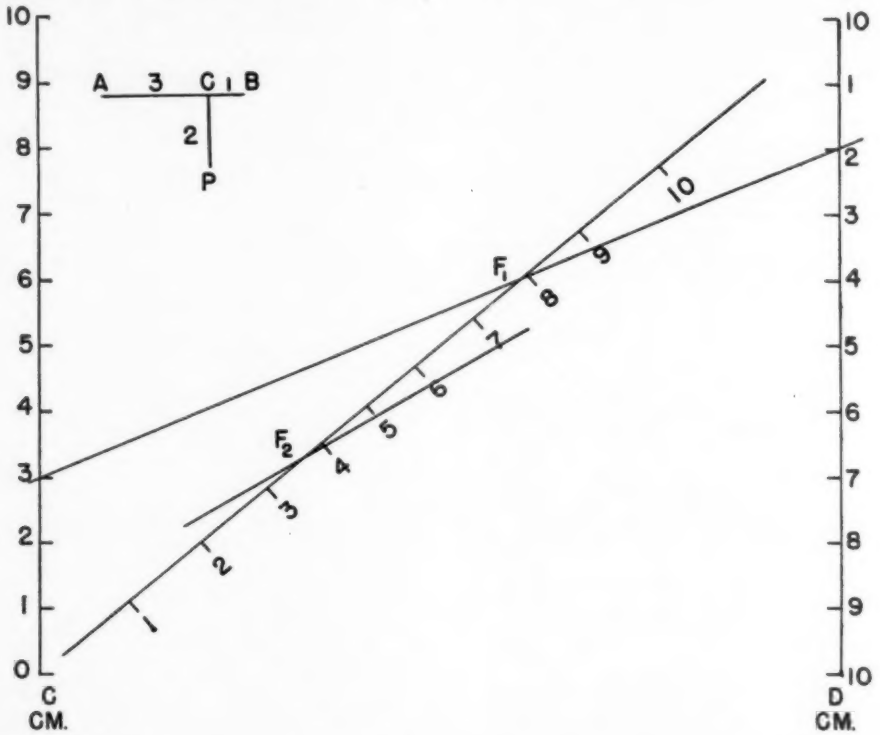

FIG. 5. Example of use of nomogram. The straight edge for the F_2 value connecting 1 on the left (*C*) scale with 2 on the right (*D*) scale is only partially shown. F_1 is derived from segment *AC*; F_2 is derived from segment *BC*.

To demonstrate the use of the nomogram, the same examples (in different order) will be solved.

Example 3 (Fig. 5). For the 3 cm. segment, we connect 3 (active length in centimeters) on the *C* scale with 2 (treatment distance in centimeters) on the *D* scale and obtain 7.8 as the first *F* value, or $F_1 = 7.8$.

For the 1 cm. segment, we connect 1 on the C scale with 2 on the *D* scale and obtain $F_2 = 3.6$. The two *F* figures are then added:

$$F_1 + F_2 = 11.4$$

This figure must be divided by 4 (active length of original or true linear source) and by 2 (treatment distance) to obtain the dose per milligram-hour:

Dose at *P* from *AB* per milligram-hour

$$= \frac{11.4}{4 \times 2} = \frac{11.4}{8} = 1.43 \text{ r}_\gamma$$

Example 4. One segment is 5 cm. long, the other 1 cm. long.

TABLE I

ROENTGENS PER MILLIGRAM-HOUR ALONG "END" AXIS; 0.5 MM. Pt

Distance in Centimeters	Active Length in Centimeters								
	.3	.4	.5	.6	.7	.8	.9	1.0	1.1
.3	72	63	55	49	44	40	36	33	31
.4	44	41	37	34	31	28.4	26.2	24.3	22.5
.5	29.5	28.5	26.3	23.8	21.7	20.0	18.5	17.2	16.2
.6	20.5	19.5	18.5	17.3	16.2	15.2	14.3	13.5	12.7
.7	15.9	15.6	15.0	14.0	13.0	12.2	11.5	10.8	10.3
.8	11.7	11.5	11.3	10.8	10.3	9.8	9.4	8.9	8.5
.9	9.3	9.2	9.0	8.8	8.5	8.2	7.8	7.5	7.2
1.0	7.5	7.5	7.5	7.4	7.2	6.9	6.6	6.4	6.2
1.2	5.3	5.3	5.3	5.2	5.1	5.0	4.8	4.7	4.5
1.4	3.9	3.9	3.9	3.9	3.8	3.7	3.6	3.6	3.5
1.6	3.0	3.0	3.0	2.95	2.92	2.88	2.84	2.80	2.75
1.8	2.40	2.40	2.40	2.38	2.36	2.33	2.30	2.27	2.25
2.0	1.95	1.95	1.95	1.94	1.93	1.91	1.90	1.88	1.87
2.2	1.62	1.62	1.62	1.62	1.59	1.58	1.56	1.55	1.54
2.4	1.36	1.36	1.36	1.36	1.34	1.33	1.32	1.30	1.29
2.6	1.16	1.16	1.16	1.15	1.15	1.14	1.13	1.12	1.11
2.8	1.00	1.00	1.00	1.00	1.00	1.00	.99	.98	.98
3.0	.88	.88	.88	.88	.88	.88	.88	.87	.86
3.2	.79	.79	.79	.79	.79	.78	.78	.77	.76
3.4	.71	.71	.71	.71	.71	.69	.69	.68	.68
3.6	.64	.64	.64	.64	.64	.63	.62	.61	.61
3.8	.56	.56	.56	.56	.56	.56	.55	.55	.54
4.0	.50	.50	.50	.50	.50	.50	.49	.49	.49
4.5	.39	.39	.39	.39	.39	.39	.39	.39	.39
5.0	.33	.33	.33	.33	.33	.33	.33	.32	.32
5.5	.270	.270	.270	.270	.270	.270	.270	.269	.268
6.0	.225	.225	.225	.225	.225	.225	.225	.224	.223
6.5	.185	.185	.185	.185	.185	.185	.185	.185	.185
7.0	.165	.165	.165	.165	.165	.165	.165	.165	.165
7.5	.140	.140	.140	.140	.140	.140	.140	.140	.140
8.0	.126	.126	.126	.126	.126	.126	.126	.126	.126

	1.2	1.3	1.4	1.5	1.6	1.7	1.8	1.9	2.0
.3	28.5	26.8	25.0	23.6	22.3	21.0	20.0	19.0	18.0
.4	21.1	19.8	18.5	17.5	16.5	15.6	14.9	14.1	13.5
.5	15.2	14.3	13.5	12.8	12.2	11.7	11.2	10.8	10.3
.6	12.0	11.4	10.8	10.3	9.8	9.4	9.0	8.6	8.2
.7	9.7	9.2	8.8	8.4	8.0	7.7	7.4	7.2	6.9
.8	8.1	7.8	7.4	7.1	6.8	6.6	6.4	6.0	5.8
.9	6.9	6.6	6.3	6.0	5.8	5.6	5.4	5.2	5.0
1.0	5.9	5.7	5.5	5.3	5.1	4.9	4.7	4.6	4.4

TABLE I—*Cont.*

Distance in Centimeters	Active Length in Centimeters								
	1.2	1.3	1.4	1.5	1.6	1.7	1.8	1.9	2.0
1.2	4.4	4.3	4.2	4.0	3.9	3.8	3.7	3.5	3.4
1.4	3.4	3.3	3.2	3.1	3.0	2.93	2.85	2.76	2.70
1.6	2.70	2.65	2.60	2.55	2.49	2.43	2.37	2.32	2.27
1.8	2.22	2.18	2.14	2.10	2.06	2.02	1.97	1.93	1.89
2.0	1.86	1.84	1.81	1.78	1.75	1.72	1.69	1.65	1.62
2.2	1.52	1.50	1.48	1.46	1.44	1.42	1.40	1.38	1.35
2.4	1.28	1.27	1.26	1.25	1.24	1.22	1.21	1.19	1.18
2.6	1.10	1.09	1.08	1.07	1.06	1.05	1.04	1.03	1.02
2.8	.97	.96	.95	.94	.93	.92	.92	.90	.90
3.0	.86	.84	.84	.83	.82	.81	.80	.80	.79
3.2	.76	.75	.75	.74	.73	.72	.72	.71	.71
3.4	.68	.67	.67	.66	.66	.65	.64	.64	.63
3.6	.60	.60	.60	.59	.59	.58	.58	.57	.57
3.8	.54	.53	.53	.53	.52	.52	.51	.51	.51
4.0	.49	.48	.48	.48	.47	.47	.46	.46	.46
4.5	.39	.39	.39	.39	.39	.39	.39	.38	.38
5.0	.32	.32	.32	.32	.31	.31	.31	.31	.31
5.5	.267	.266	.265	.265	.264	.263	.262	.261	.260
6.0	.222	.221	.220	.220	.220	.219	.218	.217	.216
6.5	.185	.185	.185	.184	.184	.184	.183	.183	.182
7.0	.164	.164	.164	.164	.163	.163	.163	.162	.162
7.5	.140	.140	.140	.140	.140	.140	.140	.140	.140
8.0	.126	.126	.126	.126	.126	.126	.126	.125	.125
	2.1	2.2	2.3	2.4	2.5	2.6	2.7	2.8	2.9
.3	17.2	16.4	15.7	15.1	14.5	14.0	13.5	13.0	12.5
.4	12.9	12.3	11.7	11.3	10.9	10.5	10.1	9.7	9.4
.5	9.9	9.5	9.1	8.8	8.5	8.2	7.9	7.6	7.4
.6	8.0	7.7	7.4	7.2	6.9	6.6	6.4	6.2	6.0
.7	6.6	6.4	6.2	6.0	5.8	5.6	5.4	5.2	5.1
.8	5.6	5.5	5.3	5.1	4.9	4.8	4.7	4.5	4.4
.9	4.8	4.7	4.5	4.4	4.3	4.1	4.0	3.9	3.8
1.0	4.3	4.2	4.0	3.9	3.8	3.7	3.6	3.5	3.4
1.2	3.3	3.3	3.2	3.1	2.99	2.90	2.83	2.75	2.68
1.4	2.63	2.56	2.50	2.44	2.38	2.32	2.27	2.22	2.17
1.6	2.21	2.15	2.10	2.05	2.00	1.95	1.91	1.86	1.83
1.8	1.85	1.81	1.77	1.73	1.69	1.65	1.62	1.59	1.56
2.0	1.59	1.56	1.53	1.50	1.47	1.45	1.42	1.39	1.37
2.2	1.33	1.31	1.29	1.27	1.25	1.23	1.21	1.19	1.17
2.4	1.16	1.14	1.13	1.11	1.09	1.07	1.05	1.04	1.02
2.6	1.01	1.00	.98	.98	.96	.94	.93	.92	.90
2.8	.89	.88	.87	.86	.85	.84	.83	.82	.81
3.0	.78	.77	.76	.76	.75	.74	.73	.72	.71

Table I—*Cont.*

Distance in Centimeters	Active Length in Centimeters								
	2.1	2.2	2.3	2.4	2.5	2.6	2.7	2.8	2.9
3.2	.70	.69	.69	.68	.67	.66	.66	.65	.64
3.4	.63	.62	.62	.61	.60	.60	.59	.58	.58
3.6	.56	.56	.56	.55	.54	.54	.53	.53	.52
3.8	.50	.50	.50	.49	.49	.49	.48	.48	.47
4.0	.45	.45	.45	.44	.44	.44	.43	.43	.43
4.5	.38	.38	.38	.37	.37	.37	.37	.37	.36
5.0	.31	.31	.30	.30	.30	.30	.299	.297	.295
5.5	.260	.259	.258	.257	.256	.255	.243	.253	.252
6.0	.216	.215	.215	.215	.215	.214	.213	.212	.211
6.5	.182	.182	.182	.182	.182	.182	.182	.182	.181
7.0	.162	.161	.161	.161	.161	.160	.160	.160	.160
7.5	.140	.140	.140	.140	.140	.140	.139	.139	.138
8.0	.125	.125	.125	.125	.125	.124	.124	.124	.123
	3.0	3.2	3.4	3.6	3.8	4.0	4.2	4.4	4.6
.3	12.0	11.2	10.5	9.8	9.3	8.8	8.4	8.0	7.6
4	9.0	8.4	8.0	7.5	7.1	6.7	6.4	6.0	5.7
5	7.1	6.7	6.4	6.0	5.7	5.4	5.1	4.9	4.6
6	5.8	5.5	5.3	5.0	4.7	4.5	4.3	4.1	3.9
.7	4.9	4.7	4.4	4.2	4.0	3.8	3.6	3.5	3.3
.8	4.3	4.0	3.8	3.6	3.5	3.3	3.2	3.0	2.89
.9	3.7	3.5	3.3	3.1	3.0	2.85	2.75	2.63	2.54
1.0	3.3	3.1	2.95	2.80	2.68	2.56	2.45	2.35	2.26
1.2	2.60	2.50	2.37	2.26	2.16	2.08	2.00	1.92	1.85
1.4	2.12	2.03	1.95	1.86	1.80	1.73	1.66	1.60	1.55
1.6	1.78	1.70	1.64	1.57	1.51	1.45	1.40	1.35	1.31
1.8	1.53	1.46	1.40	1.35	1.30	1.25	1.22	1.17	1.14
2.0	1.34	1.29	1.24	1.19	1.15	1.10	1.07	1.04	1.00
2.2	1.15	1.12	1.08	1.04	1.00	.97	.94	.91	.88
2.4	1.00	.98	.95	.92	.89	.86	.84	.82	.79
2.6	.89	.86	.84	.82	.80	.77	.75	.73	.71
2.8	.80	.78	.76	.74	.72	.70	.68	.66	.64
3.0	.70	.68	.67	.66	.64	.62	.60	.59	.57
0.2	.64	.62	.60	.59	.57	.56	.55	.53	.52
3.4	.57	.56	.54	.53	.52	.51	.50	.49	.47
3.6	.52	.51	.50	.48	.47	.46	.45	.44	.43
3.8	.47	.46	.45	.44	.43	.42	.42	.41	.40
4.0	.42	.42	.41	.40	.40	.39	.38	.38	.37
4.5	.36	.36	.35	.35	.34	.34	.33	.32	.32
5.0	.295	.293	.290	.287	.284	.280	.276	.273	.269
5.5	.251	.250	.247	.244	.241	.238	.235	.232	.229
6.0	.210	.209	.207	.205	.203	.201	.200	.197	.195

TABLE I—*Cont.*

Distance in Centimeters	Active Length in Centimeters								
	3.0	3.2	3.4	3.6	3.8	4.0	4.2	4.4	4.6
6.5	.180	.180	.180	.179	.177	.176	.175	.173	.171
7.0	.160	.158	.157	.157	.155	.155	.153	.152	.151
7.5	.138	.138	.137	.136	.135	.134	.133	.132	.131
8.0	.123	.122	.121	.120	.120	.119	.118	.117	.116
	4.8	5.0	5.2	5.4	5.6	5.8	6.0	6.2	6.4
.3	7.2	6.9	6.6	6.3	6.0	5.7	5.4	5.2	5.0
.4	5.5	5.2	5.0	4.8	4.6	4.4	4.2	4.0	3.9
.5	4.4	4.3	4.1	3.9	3.8	3.6	3.5	3.4	3.3
.6	3.7	3.6	3.5	3.3	3.2	3.1	2.95	2.85	2.75
.7	3.2	3.1	2.95	2.85	2.75	2.65	2.56	2.47	2.40
.8	2.78	2.66	2.57	2.48	2.40	2.31	2.24	2.16	2.10
.9	2.45	2.35	2.27	2.19	2.11	2.05	1.98	1.92	1.87
1.0	2.18	2.10	2.03	1.96	1.90	1.85	1.79	1.73	1.67
1.2	1.78	1.71	1.65	1.60	1.55	1.50	1.45	1.41	1.37
1.4	1.49	1.44	1.39	1.34	1.30	1.26	1.23	1.19	1.16
1.6	1.26	1.22	1.18	1.15	1.12	1.09	1.06	1.03	.99
1.8	1.10	1.06	1.03	1.00	.98	.95	.92	.90	.87
2.0	.97	.94	.91	.88	.86	.84	.82	.80	.77
2.2	.86	.83	.81	.79	.77	.75	.73	.71	.69
2.4	.77	.75	.73	.71	.70	.68	.66	.64	.63
2.6	.69	.67	.66	.64	.62	.61	.59	.58	.56
2.8	.63	.61	.60	.58	.57	.55	.54	.53	.52
3.0	.56	.55	.53	.52	.51	.50	.49	.48	.47
3.2	.51	.50	.49	.48	.47	.46	.45	.44	.43
3.4	.46	.46	.45	.44	.43	.42	.41	.40	.40
3.6	.43	.42	.41	.40	.39	.39	.38	.37	.36
3.8	.39	.38	.38	.36	.36	.36	.35	.34	.34
4.0	.36	.36	.35	.34	.34	.33	.33	.32	.32
4.5	.31	.30	.30	.295	.290	.285	.280	.275	.270
5.0	.265	.261	.257	.253	.249	.245	.241	.237	.233
5.5	.226	.223	.220	.217	.214	.211	.208	.205	.202
6.0	.193	.190	.188	.186	.184	.182	.180	.177	.175
6.5	.170	.168	.166	.164	.162	.160	.158	.157	.155
7.0	.150	.148	.147	.146	.144	.143	.141	.140	.138
7.5	.130	.129	.128	.126	.125	.124	.123	.122	.120
8.0	.115	.115	.114	.113	.112	.111	.110	.109	.108
	6.6	6.8	7.0	7.2	7.4	7.6	7.8	8.0	8.5
.3	4.8	4.6	4.4	4.2	4.0	3.9	3.8	3.7	3.3
.4	3.8	3.6	3.5	3.4	3.2	3.1	3.0	2.95	2.75
.5	3.2	3.1	2.95	2.85	2.76	2.68	2.60	2.53	2.37

TABLE I—*Cont.*

Distance in Centimeters	Active Length in Centimeters								
	6.6	6.8	7.0	7.2	7.4	7.6	7.8	8.0	8.5
.6	2.66	2.58	2.50	2.42	2.36	2.28	2.21	2.15	2.02
.7	2.32	2.25	2.20	2.13	2.06	2.01	1.95	1.90	1.80
.8	2.04	1.97	1.91	1.85	1.81	1.76	1.72	1.67	1.58
.9	1.81	1.76	1.70	1.66	1.62	1.58	1.55	1.50	1.42
1.0	1.63	1.58	1.53	1.49	1.45	1.42	1.38	1.35	1.28
1.2	1.34	1.30	1.27	1.23	1.20	1.17	1.14	1.11	1.05
1.4	1.13	1.09	1.07	1.04	1.02	.99	.96	.94	.90
1.6	.97	.94	.92	.90	.88	.86	.84	.82	.77
1.8	.85	.83	.81	.79	.77	.75	.74	.72	.68
2.0	.75	.74	.72	.70	.69	.67	.66	.64	.61
2.2	.68	.66	.64	.63	.62	.60	.59	.57	.55
2.4	.61	.60	.59	.57	.56	.55	.54	.52	.50
2.6	.55	.54	.53	.52	.50	.49	.48	.47	.45
2.8	.50	.49	.48	.47	.46	.45	.44	.43	.41
3.0	.46	.45	.44	.43	.42	.41	.40	.40	.38
3.2	.42	.41	.40	.40	.39	.38	.37	.37	.35
3.4	.39	.38	.37	.37	.36	.35	.35	.34	.33
3.6	.36	.35	.35	.34	.33	.33	.32	.32	.31
3.8	.33	.33	.32	.32	.31	.30	.30	.295	.285
4.0	.31	.30	.30	.295	.291	.287	.283	.279	.267
4.5	.265	.260	.255	.251	.247	.243	.239	.235	.225
5.0	.229	.225	.222	.218	.215	.211	.208	.204	.196
5.5	.199	.196	.193	.190	.187	.185	.183	.181	.173
6.0	.173	.170	.168	.166	.164	.162	.160	.157	.152
6.5	.153	.151	.149	.147	.145	.144	.142	.140	.135
7.0	.136	.135	.133	.131	.130	.128	.127	.125	.121
7.5	.119	.118	.116	.115	.114	.113	.112	.110	.108
8.0	.107	.106	.105	.104	.103	.102	.101	.100	.097

	9.0	9.5	10.0
.3	3.0	2.79	2.53
.4	2.55	2.40	2.23
.5	2.20	2.19	1.95
.6	1.88	1.78	1.67
.7	1.68	1.60	1.50
.8	1.49	1.40	1.33
.9	1.34	1.27	1.20
1.0	1.20	1.15	1.08
1.2	1.00	.94	.90
1.4	.84	.80	.76
1.6	.74	.70	.66
1.8	.64	.62	.59
2.0	.58	.55	.53

TABLE I—*Cont.*

Distance in Centimeters	Active Length in Centimeters		
	9.0	9.5	10.0
2.2	.52	.50	.47
2.4	.48	.46	.43
2.6	.43	.41	.39
2.8	.39	.38	.36
3.0	.36	.35	.33
3.2	.34	.32	.31
3.4	.31	.30	.290
3.6	.293	.284	.272
3.8	.274	.265	.255
4.0	.258	.250	.241
4.5	.215	.207	.199
5.0	.188	.181	.174
5.5	.167	.160	.154
6.0	.146	.140	.135
6.5	.130	.126	.122
7.0	.117	.114	.110
7.5	.105	.102	.098
8.0	.094	.091	.088

From the nomogram,

$$F_1 = 9.4; \; F_2 = 3.6$$
$$F_1 - F_2 = 9.4 - 3.6 = 5.8$$

Dose per milligram-hour from AB at

$$Q = \frac{5.8}{4 \times 2} = \frac{5.8}{8} = 0.73 \text{ r}_\gamma.$$

Example 1. For points on an end axis, there is only one "segment," i.e., the linear source itself.

$$F_1 = 8.9$$

Dose per milligram-hour at $N = \frac{8.9}{4 \times 2}$

$= 1.11 \text{ r}_\gamma$.

Example 2. For points on a central axis, the two segments are equal to each other and have the same F value.

$$F = 6.5$$
$$2F = 13.0$$

Dose per milligram-hour at $M = \frac{13.0}{4 \times 2}$

$= 1.63 \text{ r}_\gamma$.

For equivalent filtrations other than 0.5 mm. Pt, correction factors must be applied. These factors are given in Table II as percentages of the values calculated for 0.5 mm. Pt. The correction factor depends to some extent on the ratio of the distance to the active length. Two correction factors are therefore given for each filtration in order to cover the full range with an error less than 4 per cent.

When the linear source is inserted into

TABLE II

FILTRATION CORRECTION FACTORS

1 mm. Pt	1.5 mm. Pt	2.0 mm. Pt	2.5 mm. Pt
Ratio $\geq \frac{1}{4}$ 90%	Ratio $\geq \frac{1}{2}$ 80%	Ratio $\geq \frac{1}{2}$ 70%	Ratio $\geq \frac{1}{2}$ 64%
Ratio $< \frac{1}{4}$ 85%	Ratio $< \frac{1}{2}$ 75%	Ratio $< \frac{1}{2}$ 65%	Ratio $< \frac{1}{2}$ 57%

"Ratio" refers to the ratio of the perpendicular distance between the point and the source to the active length of the source.

a small volume of tissue, an additional correction must be made for tissue absorption and scattering. An approximate correction can be made by subtracting from the air dose 3 per cent for each centimeter of the distance.

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THE AMERICAN JOURNAL OF ROENTGENOLOGY AND RADIUM THERAPY

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Publisher: CHARLES C THOMAS, 220 East Monroe Street, Springfield, Illinois.

Issued Monthly. Subscription $10.00 per year, $11.00 in Canada and $12.00 in foreign countries. Advertising rates submitted on application: Editorial office, 110 Professional Building, Detroit, Mich., Office of publication, 220 E. Monroe Street, Springfield, Ill. Information of interest to all readers will be found on page iv.

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Twenty-eighth Annual Meeting: 1944, to be announced.

EDITORIAL

AN EVALUATION OF THE ROENTGEN METHODS USED IN MASS CHEST SURVEYS

IT IS generally recognized that the incidence of pulmonary tuberculosis might be effectively reduced if those individuals with active disease were isolated at the earliest possible moment. Since early isolation presupposes early detection and since the roentgen examination constitutes the most reliable method for the early diagnosis of pulmonary tuberculosis, it is evident that considerable progress in the control of this disease might be achieved if the population at large were examined roentgenographically at regular intervals. Until the early 1930's such a program was impractical due to the excessive cost of the roentgen methods then available. In recent years, however, this situation has been spectacularly changed by the development of the photofluorographic process in which a fluorescent image of the chest is photographed on films of reduced size (usually 4×5 inch, or 35 mm. film). Such films, when made in large numbers, are considerably less expensive to produce, process and handle than standard 14×17 inch roentgenograms. Recently, the roll paper process in which 14×17 inch roentgenograms are made on sensitized paper has been perfected and may also prove a useful method for the examination of large population groups.

The photofluorographic process has been used extensively in this country in the examination of inductees entering the armed services and of many thousands of civilians engaged in industry, and there is every reason to expect that the process will receive progressively wider application as time goes on. Before the outbreak of the current war, photofluorography was an established procedure in many foreign countries, particularly in Brazil and Scandinavia.

The diagnostic quality of photofluorographic and paper film is somewhat inferior to that of 14×17 inch roentgenograms and, as a result, many radiologists have become concerned lest the diagnostic error of these newer methods be high and thereby largely counteract their other advantages. In an effort to determine the relative merits of the various roentgen methods which are available for the examination of the chest (14×17 inch roentgenographic film, 4×5 inch photofluorographic film, 35 mm. photofluorographic film, 14×17 inch sensitized paper, and roentgenoscopy), clinical studies in which two or more of the methods were compared have been made by several investigators. The conclusions reached vary widely and seem to depend on the experience or inexperience, as the case may be, of the interpreter. Furthermore, many of these studies have not been carefully controlled from the viewpoint of selection of samples and statistical analysis and accordingly the overall picture tends to be confused.

A direct quantitative evaluation of the diagnostic quality of the various roentgen procedures may be made by a comprehensive investigation of the physical characteristics of the several methods. From such a study it is possible to list in unequivocal terms the relative detail with which roentgen images may be resolved by each method and to determine the smallest lesion which a given method is capable of recording. During the past two years such a study has been under way at the University of Chicago. An attempt has been made to correlate the various physical factors which

influence roentgenographic and roentgenoscopic quality so that the part played by each in the total effect may be correctly evaluated. Physiological considerations of viewing have also been included. Although this material will be presented in detail in a series of articles which will appear in forthcoming issues of this JOURNAL, it has been thought desirable to summarize here some of the preliminary findings of the study so far as they relate to the various roentgen methods used in the examination of the chest.

The resolving power of 14×17 inch roentgenograms is superior to that of all other roentgenographic materials. In order of merit below these films may be listed, 4×5 inch photofluorograms, when made with a stationary-focused grid; 14×17 inch sensitized paper; 35 mm. photofluorograms when made with a stationary-focused grid, and 4×5 inch photofluorograms, made without a grid; 35 mm. photofluorograms, made without a grid; and, finally, roentgenoscopy.

The low resolution provided by the roentgenoscopic method may be attributed to the low visual acuity of the eye when operating at the low intensity levels present under roentgenoscopic conditions. A grid improves the diagnostic quality of photofluorograms by reducing the amount of scattered radiation reaching the fluorescent screen, and thereby increasing the contrast of the roentgen images. Due to the high inherent contrast of 14×17 inch roentgenographic film, and the relatively short useful density range of 14×17 inch paper film, a grid is not useful when these materials are employed.

It must be realized that the above list represents conditions as of 1944, and undoubtedly it will require considerable revision from time to time in the years to come. Indeed, there is reason to expect that, before long, 4×5 inch photofluorograms and eventually 35 mm. photofluorograms will be equivalent to present-day 14×17 inch roentgenograms.

Although the foregoing discussion indicates the relative merits of the various roentgen procedures used in the examination of the chest, it does not provide any information as to the diagnostic error which may be expected from a given method in the detection of pulmonary tuberculosis. Physical studies reveal, however, that the smallest infiltrative lesion which may be detected by a competent observer using roentgenoscopy is between 0.5 and 1.0 cm. in diameter; smaller lesions, of course, can be detected with the other methods, with 14×17 inch roentgenograms being able to reproduce the smallest process. Since most significant lesions exceed 0.5 to 1.0 cm. in diameter, it is evident that all of the roentgen methods which are available for the examination of the chest should be capable of detecting the majority of tuberculous processes. One word of caution must be injected at this point, however. Since the resolution or detail with which a lesion may be seen varies widely in the different methods, one must not use the same diagnostic standards when interpreting the films produced by one method that are used when interpreting the films produced by another. For example, a particular tuberculous lesion will present considerably less detail in a 35 mm. film than that seen in a 14×17 inch roentgenogram. Indeed, if the lesion is small, its true significance may be easily overlooked if the roentgenologist unconsciously insists on the fulfillment of the criteria employed in the diagnosis of the larger film. A spectacular instance of this occurred recently when an acquaintance of the writer was asked to test his skill in interpreting 35 mm. photofluorograms. Fourteen by seventeen inch and 35 mm. films of several hundred individuals, of whom approximately 20 per cent were known to have minimal pulmonary tuberculosis, were submitted to this well known radiologist who until this time had read relatively few small films. The results of the test were at first highly disconcerting because a considerable number of cases called "positive" from the 14×17 inch films were called "negative" from the 35 mm. films. For-

tunately, however, the radiologist did not stop here but wisely reviewed the two series of films, comparing each 35 mm. photofluorogram with each 14×17 inch roentgenogram. In this way, he was able to establish for himself the diagnostic criteria which should be used in the interpretation of small films. After this educational program, another comparative series of 35 mm. photofluorograms and 14×17 inch roentgenograms was submitted to him for interpretation. The diagnostic error in reading the small films this time was of a much lower order.

The foregoing is a forceful demonstration that an ability to correctly diagnose the films produced by one roentgenographic method does not presuppose an equal ability to interpret the films produced by another. First, it is necessary for one to establish for himself the diagnostic criteria which are more or less peculiar to the roentgenograms made by a particular method. Such education may be accomplished in at least two ways. In one, perspective may be gained by the examination of many thousands of films observed over a considerable period of time. This is the method which almost every roentgenologist employs during his training period. The trained roentgenologist, however, may follow a less tedious procedure. He may establish the diagnostic criteria which he is seeking by comparing the appearance of a representative number of tuberculous lesions produced on films with which he is familiar (for example, 14×17 inch roentgenograms) with those produced on films with which he is unfamiliar. Such a method was used by the roentgenologist referred to above.

In summarizing, it is evident that all of the newer roentgenological methods which are in use today can be considered acceptable from a diagnostic standpoint for use in mass roentgen surveys for tuberculosis control. It is extremely important, however, that the interpreter of the films clearly understands the diagnostic criteria of the roentgenographic process which is employed. Failure to do this will immediately introduce a large number of diagnostic errors.

R. H. M.

SOCIETY PROCEEDINGS, CORRESPONDENCE AND NEWS ITEMS

Items for this section solicited promptly after the events to which they refer.

MEETINGS OF ROENTGEN SOCIETIES*

UNITED STATES OF AMERICA

AMERICAN ROENTGEN RAY SOCIETY
Secretary, Dr. H. Dabney Kerr, University Hospital, Iowa City, Iowa. Annual meeting: Joint Meeting of American Roentgen Ray Society and Radiological Society of North America, Palmer House, Chicago, Ill., Sept. 24–29, 1944.

AMERICAN COLLEGE OF RADIOLOGY
Secretary, Mac F. Cahal, 540 N. Michigan Ave., Chicago, Ill. Annual Meeting: Stevens Hotel, 10:00 A.M., June 14, 1944.

SECTION ON RADIOLOGY, AMERICAN MEDICAL ASSOCIATION
Secretary, Dr. J. T. Murphy, 421 Michigan St., Toledo, Ohio. Annual meeting: Chicago, Ill., June 12–16, 1944.

ARKANSAS RADIOLOGICAL SOCIETY
Secretary, Dr. J. S. Wilson, Mack Wilson Hospital, Monticello, Ark. Meets every three months and also at time and place of State Medical Association.

RADIOLOGICAL SOCIETY OF NORTH AMERICA
Secretary, Dr. D. S. Childs, 607 Medical Arts Bldg., Syracuse, N. Y. Annual meeting: Joint Meeting of American Roentgen Ray Society and Radiological Society of North America, Palmer House, Chicago, Ill., Sept. 24–29, 1944.

RADIOLOGICAL SECTION, BALTIMORE MEDICAL SOCIETY
Secretary, Dr. Walter L. Kilby, Baltimore. Meets third Tuesday each month, September to May.

SECTION ON RADIOLOGY, CALIFORNIA MEDICAL ASSOCIATION
Secretary, Dr. Earl R. Miller, University of California Hospital, San Francisco, Calif.

RADIOLOGICAL SECTION, CONNECTICUT MEDICAL SOCIETY
Secretary, Dr. Max Climan, 242 Trumbull St., Hartford, Conn. Meets bi-monthly on second Thursday, at place selected by Secretary. Annual meeting in May.

SECTION ON RADIOLOGY, ILLINOIS STATE MEDICAL SOCIETY
Secretary, Dr. H. W. Ackemann, 321 W. State St., Rockford, Ill.

RADIOLOGICAL SECTION, LOS ANGELES COUNTY MEDICAL ASSOCIATION
Secretary, Dr. Roy W. Johnson, 1407 S. Hope St., Los Angeles, Calif. Meets on second Wednesday of each month at the County Society Building.

RADIOLOGICAL SECTION, SOUTHERN MEDICAL ASSOCIATION
Secretary, Dr. Roy G. Giles, Temple, Texas.

BROOKLYN ROENTGEN RAY SOCIETY
Secretary, Dr. Leo Harrington, 880 Ocean Ave., Brooklyn, N.Y. Meets monthly on fourth Tuesday, October to April.

BUFFALO RADIOLOGICAL SOCIETY
Secretary, Dr. Joseph S. Gian-Francheschi, 610 Niagara St., Buffalo, N. Y. Meets second Monday of each month except during summer months.

CHICAGO ROENTGEN SOCIETY
Secretary, Dr. F. H. Squire, 1754 W. Congress St., Chicago 12, Ill. Meets second Thursday of each month October to April inclusive at the Palmer House.

CINCINNATI RADIOLOGICAL SOCIETY
Secretary, Dr. Samuel Brown, 707 Race St., Cincinnati, Ohio. Meets third Tuesday of each month, October to May, inclusive.

CLEVELAND RADIOLOGICAL SOCIETY
Secretary, Dr. D. D. Brannan, 11311 Shaker Blvd., Cleveland 4, Ohio. Meets at 6:30 P.M. at Allerton Hotel on fourth Monday each month, October to April, inclusive.

DALLAS-FORT WORTH ROENTGEN STUDY CLUB
Secretary, Dr. X. R. Hyde, Medical Arts Bldg., Fort Worth, Texas. Meetings held in Dallas on odd months and in Fort Worth on even months, on third Monday, at 7:30 P.M.

DENVER RADIOLOGICAL CLUB
Secretary, Dr. Edward J. Meister, 366 Metropolitan Bldg., Denver, Colo. Meets third Friday of each month at Denver Athletic Club.

DETROIT ROENTGEN RAY AND RADIUM SOCIETY
Secretary, Dr. E. R. Witwer, Harper Hospital. Meets monthly on first Thursday from October to May, at Wayne County Medical Society Building.

FLORIDA RADIOLOGICAL SOCIETY
Acting Secretary, Dr. Walter A. Weed, 204 Exchange Bldg., Orlando, Fla. Meetings in May and November.

GEORGIA RADIOLOGICAL SOCIETY
Secretary, Dr. James J. Clark, 478 Peachtree St., Atlanta, Ga. Meets in November and at annual meeting of Medical Association of Georgia in the spring.

RADIOLOGICAL SOCIETY OF KANSAS CITY
Secretary, Dr. Arthur B. Smith, 800 Argyle Bldg., Kansas City, Mo. Meets third Thursday of each month at a place designated by the president.

ILLINOIS RADIOLOGICAL SOCIETY
Secretary, Dr. Wm. DeHollander, St. John's Hospital, Springfield, Ill. Meets three times a year.

INDIANA ROENTGEN SOCIETY
Secretary, Dr. H. C. Ochsner, Methodist Hospital, Indianapolis. Meeting held the second Sunday in May annually.

KENTUCKY RADIOLOGICAL SOCIETY
Secretary, Dr. W. C. Martin, 321 W. Broadway, Louisville. Meets annually in Louisville on first Saturday in Apr.

LONG ISLAND RADIOLOGICAL SOCIETY
Secretary, Dr. Marcus Wiener, 1430-48th St., Brooklyn, N. Y. Meets Kings County Med. Soc. Bldg. monthly on fourth Thursday, October to May, 8:30 P.M.

LOUISIANA RADIOLOGICAL SOCIETY
Secretary, Dr. J. R. Anderson, 1130 Louisiana Ave., Shreveport. Meets annually during Louisiana State Medical Society Meeting.

MICHIGAN ASSOCIATION OF ROENTGENOLOGISTS
Secretary, Dr. E. M. Shebesta, 1429 David Whitney Bldg., Detroit. Three meetings a year, Fall, Winter, Spring.

MILWAUKEE ROENTGEN RAY SOCIETY
Secretary, Dr. C. A. H. Fortier, 231 W. Wisconsin Ave., Milwaukee, Wis. Meets monthly on second Monday at University Club.

MINNESOTA RADIOLOGICAL SOCIETY
Secretary, Dr. Annette T. Stenstrom, 1218 Medical Arts Bldg., Minneapolis, Minn. One meeting a year at time of Minnesota State Medical Association.

NEBRASKA RADIOLOGICAL SOCIETY
Secretary, Dr. D. A. Dowell, Medical Arts Bldg., Omaha, Nebr. Meets third Wednesday of each month, at 6 P.M. at either Omaha or Lincoln.

NEW ENGLAND ROENTGEN RAY SOCIETY
Secretary, Dr. Hugh F. Hare, Lahey Clinic, Boston, Mass. Meets monthly on third Friday, Boston Medical Library.

RADIOLOGICAL SOCIETY OF NEW JERSEY
Secretary, Dr. H. R. Brindle, 501 Grand Ave., Asbury Pk. Meets annually at time and place of State Medical Society. Mid-year meetings at place chosen by president.

NEW YORK ROENTGEN SOCIETY
Secretary, Dr. Ramsay Spillman, 115 East 61st St., New York City. Meets monthly on third Monday, New York Academy of Medicine, at 8:00 P.M.

NORTH CAROLINA ROENTGEN RAY SOCIETY
Secretary, Dr. Major Fleming, Rocky Mount, N. C. Annual meeting at time and place of State Medical Society. Mid-year scientific meeting at place designated.

* Secretaries of Societies not here listed are requested to send the necessary information to the Editor.

North Dakota Radiological Society
Secretary, Dr. L. A. Nash, St. John's Hospital, Fargo. Meetings held by announcement.

Central New York Roentgen Ray Society
Secretary, Dr. C. F. Potter, 820 S. Crouse Ave., Syracuse. Three meetings a year. January, May, November.

Ohio Radiological Society
Secretary, Dr. J. E. McCarthy, 707 Race St., Cincinnati. Meets at time and place of annual meeting of Ohio State Medical Association.

Pacific Roentgen Society
Secretary, Dr. L. H. Garland, 450 Sutter St., San Francisco, Calif. Meets annually, during meeting of California Medical Association.

Pennsylvania Radiological Society
Secretary, Dr. L. E. Wurster, 416 Pine St., Williamsport. Annual meeting at Penn Alto Hotel, Altoona, Pa., May 13–14, 1944.

Philadelphia Roentgen Ray Society
Secretary, Dr. R. P. Barden, University Hospital, Meetings first Thursday of each month from October to May inclusive at 8:15 P.M., in Thompson Hall, College of Physicians, 19 S. 22d St.

Pittsburgh Roentgen Society
Secretary, Dr. R. G. Alley, 4800 Friendship Ave. Meets second Wednesday each month, 4:30 P.M., October to June, Pittsburgh Academy of Medicine.

Rochester Roentgen Ray Society, Rochester, N. Y.
Secretary, Dr. Sidney Larson, Strong Memorial Hospital. Meets monthly on third Monday from October to May, inclusive, 8 P.M. at Strong Memorial Hospital.

Rocky Mountain Radiological Society
Secretary, Dr. A. M. Popma, 220 N. First St., Boise, Idaho.

St. Louis Society of Radiologists
Secretary, Dr. E. W. Spinzig, 2646 Potomac, St. Louis, Mo. Meets fourth Wednesday of each month, except June, July, August, and September, at a place designated by the president.

San Diego Roentgen Society
Secretary, Dr. Henry L. Jaffe, Naval Hospital, Balboa Park, San Diego, Calif. Meets monthly on first Wednesday at dinner.

San Francisco Radiological Society
Secretary, Dr. Martha Mottram, 450 Sutter St., San Francisco. Meets monthly on third Thursday at 7:45 P.M., first six months of year at Toland Hall, University of California Hospital, second six months at Lane Hall, Stanford University Hospital.

Shreveport Radiological Club
Secretary, Dr. R. W. Cooper, Charity Hospital, Shreveport, La. Meets monthly on third Wednesday, at 7:30 P.M., September to May inclusive.

South Carolina X-Ray Society
Secretary, Dr. T. A. Pitts, Baptist Hospital, Columbia, S. C. Meets in Charleston on first Thursday in November, also at the time and place of South Carolina State Medical Association.

Tennessee Radiological Society
Secretary, Dr. J. M. Frère, 707 Walnut St., Chattanooga, Tenn. Meets annually at the time and place of the Tennessee State Medical Association.

Texas Radiological Society
Secretary, Dr. Herman Klapproth, Sherman, Texas.

University of Michigan Department of Roentgenology Staff Meeting
Meets each Monday evening from September to June, at 7 P.M. at University Hospital.

University of Wisconsin Radiological Conference
Secretary, Dr. E. A. Pohle, 1300 University Ave., Madison, Wis. Meets every Thursday from 4:00–5:00 P.M., Room 301, Service Memorial Institute.

Virginia Radiological Society
Secretary, Dr. E. L. Flanagan, 116 E. Franklin St., Richmond, Va. Meets annually in October.

Washington State Radiological Society
Secretary, Dr. Thomas Carlile, 1115 Terry St., Seattle. Meets fourth Monday each month, October through May, College Club, Seattle.

X-ray Study Club of San Francisco
Secretary, Dr. J. M. Robinson, University of California Hospital. Meets monthly in evening on third Thursday.

Cuba

Sociedad de Radiología y Fisioterapia de Cuba
President, Dr. J. Manuel Viamonte, Hospital Mercedes, Habana, Cuba. Meets monthly in Habana.

British Empire

British Institute of Radiology Incorporated with the Röntgen Society
Medical Members' meeting held monthly on third Friday at 2:30 P.M. and Ordinary Meeting at same time on following Saturday, October to May, 32 Welbeck St., London, W.1.

Section of Radiology of the Royal Society of Medicine (Confined to Medical Members)
Meets on the third Friday of each month at 4:45 P.M. at the Royal Society of Medicine 1, Wimpole St., London, W. 1.

Faculty of Radiologists
Secretary, Dr. M. H. Jupe, 32 Welbeck St., London, W. 1 England.

Section of Radiology and Medical Electricity, Australasian Medical Congress
Secretary, Dr. H. M. Cutler, 139 Macquarie St., Sydney, New South Wales.

Radiological Section of the Victorian Branch of the British Medical Association
Secretary, Dr. Keith Hallam, St. George's Hospital, K.E.W., Melbourne, E. 4, Victoria, Australia. Meets monthly from March to November inclusive.

Canadian Association of Radiologists
Secretary, Dr. A. D. Irvine, 540 Tegler Bldg., Edmonton, Alberta.

Section of Radiology, Canadian Medical Association
Secretary, Dr. C. M. Jones, Inglis St., Ext. Halifax, N. S.

Radiological Section, New Zealand British Medical Association
Secretary, Dr. Colin Anderson, Invercargill, New Zealand. Meets annually.

South America

Sociedad Argentina de Radiologia
Secretary, Dr. Guido Gotta, Buenos Aires, Argentina. Meetings are held monthly.

Continental Europe

Sociedad Espanola de Radiologia y Electrologia
Secretary, Dr. J. Martin-Crespo, Fuencarral, 7. Madrid, Spain. Meets monthly in Madrid.

Société Suisse de Radiologie (Schweizerische Röntgen-Gesellschaft)
Secretary for French language, Dr. A. Grosjean La Chaux de Fonds.
Secretary for German language, Dr. Scheurer, Molzgasse Biel. Meets annually in different cities.

Societatea Romana de Radiologie si Electrologie
Secretary, Dr. Oscar Meller, Str. Banul Mărăcine, 30, S. I., Bucuresti, Roumania. Meets second Monday in every month with the exception of July and August.

All-Russian Roentgen Ray Association, Leningrad: USSR in the State Institute of Roentgenology and Radiology, 6 Roentgen St.
Secretaries, Drs. S. A. Reinberg and S. G. Simonson. Meets annually.

Leningrad Roentgen Ray Society
Secretaries, Drs. S. G. Simonson and G. A. Gusterin. Meets monthly, first Monday at 8 o'clock, State Institute of Roentgenology and Radiology, Leningrad.

Moscow Roentgen Ray Society
Secretaries, Drs. L. L. Holst, A. W. Ssamygin and S. T. Konobejevsky. Meets monthly, first Monday, 8 P.M.

Scandinavian Roentgen Societies
The Scandinavian roentgen societies have formed a joint association called the Northern Association for Medical Radiology, meeting every second year in the different countries belonging to the Association.

JOINT MEETING OF THE AMERICAN ROENTGEN RAY SOCIETY AND THE RADIOLOGICAL SOCIETY OF NORTH AMERICA

Attention is again called to the Joint Meeting of the American Roentgen Ray Society and the Radiological Society of North America which is to be held at the Palmer House, Chicago, Illinois, September 24 to 29, 1944. Plans are progressing satisfactorily and a very successful meeting is foreseen. Committees have been appointed which are looking after all of the details in connection with the meeting.

The Coordinating Committee consists of Dr. L. C. Kinney, 1831 Fourth Avenue, San Diego 1, California, Dr. E. R. Witwer, Harper Hospital, Detroit 1, Michigan, and Dr. E. L. Jenkinson, St. Luke's Hospital, Chicago, Illinois. Titles and abstracts of papers should be sent to Dr. Kinney or Dr. Witwer before July 1, 1944.

Dr. Lawrence Reynolds, 110 Professional Building, Detroit 1, Michigan, is Chairman of the Committee on Commercial Exhibits. Those who have not already taken space in the exhibit and are interested in doing so should communicate with Dr. Reynolds at an early date.

The Committee on Scientific Exhibits of which Dr. Clarence E. Hufford, 421 Michigan Street, Toledo, Ohio, is Chairman anticipates assembling an instructive and interesting exhibit.

Dr. Warren W. Furey, 6844 Oglesby Avenue, Chicago, Illinois, is in charge of the Refresher Courses which will begin on Sunday, September 24. These are always an important part of a meeting and it is certain that this year they will prove of more than ordinary interest and educational value.

The complete program of the meeting will be published in a future issue of the JOURNAL. In the meantime it is hoped that radiologists will make their plans to attend and that those who wish to take an active part in the meeting will communicate as soon as possible with those in charge of the arrangements.

RADIOLOGICAL SOCIETY OF NEW JERSEY

At the annual meeting of the Radiological Society of New Jersey held in Atlantic City on April 26, 1944, the following officers were elected for the ensuing year: *President*, Dr. J. H. Wyatt; *Vice President*, Dr. H. J. Perlberg; *Secretary*, Dr. H. R. Brindle; *Treasurer*, Dr. W. H. Seward; *Counsellor*, Dr. W. O. Wuester.

AMERICAN BOARD OF RADIOLOGY

The American Board of Radiology will conduct examinations on September 22, 23, and 24, 1944, at the Palmer House, Chicago, Illinois. Those wishing to be examined at that time must have their application on file by August 1, 1944.

B. R. KIRKLIN
Secretary

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ROENTGEN AND RADIUM THERAPY

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ABSTRACTS OF ROENTGEN AND RADIUM LITERATURE

ROENTGEN DIAGNOSIS

GENITOURINARY SYSTEM

SWEETSER, T. H. Retroperitoneal tumors influencing the kidneys and ureters. *J. Urol.*, May, 1942, *47*, 619–631.

The advent of intravenous and retrograde urography, exposing films in anteroposterior and lateral projections, has helped in the diagnosis of retroperitoneal tumors, but the diagnosis may still be difficult. The authors present 8 case reports to illustrate some of the difficulties.

The first case was a sixteen year old boy with an abdominal mass and hematuria. A retrograde pyelogram showed upward displacement of the right kidney and mesial displacement of the right ureter; operation revealed a retroperitoneal neuro-epithelioma. The second case was that of a twenty-four year old woman with an obvious perinephric abscess. The sinus tract did not heal following drainage and a retrograde pyelogram showed the presence of the abscess and a hydronephrosis. Compression of the ureter was present, as well as forward and lateral displacement. This led to the impression of a retroperitoneal mass as the etiological factor in the whole picture. Operation revealed an undifferentiated, very malignant tumor compressing the ureter.

The third case was a boy of fifteen who complained of recurrent abdominal pain, and swelling. Roentgenographic studies showed a retroperitoneal tumor, containing calcium, which was displacing the kidney downward and the diaphragm upward. The stomach was displaced forward and to the right. A high diastolic blood pressure and periodic headaches suggested an adrenal medullary tumor. Operation disclosed an adrenal paraganglioma. The roentgen findings might have been simulated by a cyst of the tail of the pancreas.

The fourth case was that of a sixty-six year old man who complained of lumbar pain, frequency and nocturia. An abdominal mass could be felt. The right ureter was displaced mesially. Operation revealed an adenocarcinoma of the colon.

Case 5 was a twenty year old woman who complained of right flank pain. An intravenous urogram showed outward and downward displacement of the right kidney. The left ureter showed slight lateral displacement. The spleen was enlarged and several discrete cervical nodes were palpable. The diagnosis was Hodgkin's disease and the abdominal mass was due to enlarged abdominal lymph nodes. A simple renal cyst was present which added to the confusing picture and raised the question of a primary renal tumor with metastasis to the lymph nodes.

The next 2 cases showed displacement of the kidneys and ureters from enlarged lymph nodes due to metastases from primary tumors in the testicle and ureter respectively. The eighth case had a mass overlying the promontory of the sacrum which proved to be a hydronephrotic ectopic kidney.

In conclusion, roentgenography has proved to be the greatest single aid in diagnosing these confusing cases.—*R. M. Harvey.*

PRINCE, C. L. Malignant tumors of the spermatic cord; a brief review with presentation of a case of angio-endothelioma. *J. Urol.*, June, 1942, *47*, 793–799.

This case is the first angio-endothelioma of the spermatic cord and the seventy-fifth malignant tumor of the cord to be reported. The patient was a white male, aged sixty-three, who had had a progressive swelling of the scrotum for fourteen months. Growth had been rapid for three months. Physical examination revealed a mass arising from the upper pole of the testis which did not transmit light. A roentgenogram showed no calcification.

At operation the contents of the left scrotum were removed en masse and a tumor of the spermatic cord was found which histologically proved to be an angio-endothelioma. Postoperative roentgen therapy was given. Twenty-seven months later the patient returned with apparent orbital metastases which responded readily to roentgen therapy.

Seventy per cent of tumors of the spermatic cord are benign, according to Thompson; 71 of the reported 74 malignant tumors were sarcoma and only 3 carcinoma. The classification of tumors of the spermatic cord as proposed by

Hinman and Gilison is given by the author. They divide the tumors both benign and malignant into three groups: epithelial, mesoblastic and heterologous. The dermoids are included in the benign heterologous group and the teratomas in the malignant heterologous group. The angio-endotheliomata, such as reported in this article by Prince, are supposed to arise in cells lining vascular and lymphatic channels. This should serve to put these tumors in the sarcomatous group because of mesoblastic origin.

The differential diagnosis of spermatic cord tumors must include hernia, hydrocele, hematocele, spermatocele and testicular tumors.

The treatment of a malignant cord tumor is thorough removal of the tumor, testis, epididymis and as much of the cord as possible followed by postoperative deep roentgen therapy. —*R. M. Harvey.*

Jewett, Hugh J., Sloan, Louise L., and Strong, George H. Does vitamin A deficiency exist in clinical urolithiasis? *J. Am. M. Ass.*, Feb. 20, 1943, *121*, 566–569.

This investigation consisted in a clinical study of 20 patients and a microscopic examination of 78 autopsy cases with urinary calculi. In the clinical group the authors attempted to select reliable visual tests for vitamin A deficiency and have supplemented these subjective tests with the determinations of the level of vitamin A in the blood. In obtaining data, 20 patients with urolithiasis in the Brady Urological Institute and 40 normal subjects comprising a control group were given a series of tests. Determinations of the concentrations of vitamin A in the blood plasma were made in the 20 cases of urolithiasis and in 33 normal controls.

In summary, the writers say that in none of these 98 cases was there any evidence of vitamin A deficiency. Although a vitamin A free diet produces widespread epithelial changes which may lead to the formation of urinary calculi in experimental animals, there is no positive proof that a "subclinical" vitamin A deficiency is an etiologic factor in urolithiasis in man.—*S. G. Henderson.*

Thompson, G. J., and Greene, L. F. Ureterocele; a clinical study and a report of 37 cases. *J. Urol.*, June, 1942, *47*, 800–809.

Excretory urography rarely demonstrates a ureterocele although it will reveal the ureterectasis and pyelectasis which may accompany it. Most authors believe ureterocele to be congenital in origin. Gottlieb believes that there are two causative factors—an abnormally narrow ureteral orifice and a weakness of Waldeyer's sheath.

Other congential deformities are frequently associated with ureterocele which supports the congenital theory of their etiology. Some cases are undoubtedly of congenital origin, and some of acquired origin.

Two-thirds of the authors' cases occurred between the ages of thirty and fifty. The symptoms are inconstant, with pain the most frequent. Other symptoms are hematuria, frequency, dysuria, etc. The cystoscopic picture may be confusing in the presence of associated inflammation and edema.

In excretory urograms a ureterocele appears as a spherical or sausage-shaped filling defect in the bladder near the mouth of the ureter. Occasionally a surrounding halo is present. Stones may be present in the ureterocele. The authors, in a series of 21 cases studied by excretory urography, were able to make a diagnosis of ureterocele in only 4 cases on the urogram alone. The most common roentgen finding was an irregular dilatation of the lower third of the ureter which tapered to a point at the ureteral orifice. Although they did not find the excretory urogram of special value in making a specific diagnosis of ureterocele, the authors were able to demonstrate other congenital anomalies of the urinary tract, to estimate kidney function, to note secondary obstructive uropathies and to show stones within the ureteroceles.

Treatment is not necessary in the absence of symptoms. Dilatation of the ureteral orifice may be of temporary benefit. The most satisfactory form of treatment is ureteromeatotomy. The results of treatment are entirely satisfactory.—*R. M. Harvey.*

Kretschmer, Herman L. Stone in the ureter; clinical data based on 500 cases. *Surg., Gynec. & Obst.*, June, 1942, *74*, 1065–1077.

This paper is based on a study of 500 unselected patients with stone in the ureter, covering a period from 1917 to 1941.

Sex. Of the 500 patients studied, 400 were males and 100 females.

Age. Stone in the ureter is rare at the extremes of life. Stone occurred with the greatest frequency in the third, fourth, and fifth decades.

Location. The stone was located on the right side in 229 patients, on the left side in 259 and on both sides in 12.

The Stone. (1) Number of stones: In the largest number of instances the symptoms are due to the presence of a single stone, 89.4 per cent of the present series. Multiple stones may vary greatly in number from 2 to 100 or more. (2) Size: Fortunately, most stones in the ureter are small. (3) Chemical composition of stones: 102 stones were analyzed; in only 9 cases was only one element present. Calcium phosphate was the predominant element in 45 stones and calcium oxalate in 35.

Symptoms and Signs.

1. Pain was the first symptom in 88 per cent of the cases. It occurred at some time during the illness in 93.6 per cent. The pain usually radiates downward along the course of the ureter to the internal or external genitalia or upward into the renal area. The pain is usually very severe.

2. Hematuria may be the first and only symptom of which the patient complains. Hematuria was present in 34.2 per cent at some time during the illness and in 4.6 per cent as the first symptom noted.

3. Nausea and vomiting occurred in 46.2 per cent. Previous passage of stones was elicited in the history of 14.2 per cent of these patients.

4. Other symptoms. Backache was present in 37.2 per cent. Frequency of urination was noted in 42 per cent and burning in 30.4 per cent. Thirty-six per cent stated that pus had previously been found in the urine. Chills and fever were present in 17.6 per cent.

The Examination.

1. Urinalysis. The findings were positive in 83.43 per cent and negative in 15.56 per cent. The results of the urinary examinations were as follows: albumin in 24.4 per cent; sugar in 0.8 per cent; red blood cells in 43 per cent; white blood cells in 69.2 per cent; casts in 7.6 per cent. The importance of repeated examinations cannot be overemphasized.

2. Culture studies of the urine. Cultures were made in 385 patients of which 34.54 per cent were positive and 65.43 per cent were sterile.

3. Blood pressure studies. These were normal except in patients in the older age group.

4. Roentgen-ray observations. This begins with a plain roentgenogram. Positive reports were obtained in 95.79 per cent and negative reports in 4.21 per cent. The presence of a shadow along the course of the ureter warrants further investigation to determine whether one is dealing with a stone or with an extra-ureteral shadow. This can be done by the use of a shadowgraph catheter with a double exposure film and shift in the tube. In the case of very stout patients it is advantageous to use two films.

5. Intravenous urography. In most cases it reveals dilatation of the ureter and of the kidney pelvis. Stone in the ureter is probably the most frequent cause of hydronephrosis. In some instances the urogram fails to visualize the outline of the pelvis and ureter while an increased density of the kidney outline is shown. This is probably due to a temporary inhibition of function.

6. Retrograde movement of stone. Because of this possibility a roentgenogram should be made on each patient just before operation. Retrograde movement of the stone occurred in 10 patients in the present series.

Treatment. (1) One hundred and twenty-eight patients passed their stones without treatment; 196, with manipulation; and 114 were operated on. (2) Dangers of too frequent manipulation are stressed by the author. (3) A policy of "watchful waiting" in the average case is still desirable.—*Mary Frances Vastine.*

HIEMSTRA, W., and CREEVY, C. D. Tumors of the urinary bladder. *Radiology*, August, 1942, *39*, 175–183.

This article is a report of 174 cases of tumor of the bladder treated at the University Hospitals, University of Minnesota Medical School from January 1, 1930 to December 31, 1939. They included 28 benign papillomas and 146 carcinomas, 57 of which were papillary (39 per cent) and 89 infiltrating (61 per cent). Most of the carcinomas were too advanced to permit of radical operation. Tables and graphs are given showing the details of the treatment and results. Cases without metastases showed a five year survival rate of 15.7 per cent and those with metastases only 5.3 per cent. Among 70 cases of the infiltrating type the five year survival rate was only 4.2 per cent.

In the carcinoma cases the average duration of symptoms before reporting for treatment was 2.3 years. This should be materially reduced by educating the public in regard to the early symptoms of carcinoma of the bladder and by bringing about better cooperation between the public and the physicians. Hematuria is the commonest first symptom and the public does not realize its seriousness. They should be

taught to report for examination on the appearance of the slightest degree of hematuria. Increased frequency and dysuria were the next most frequent symptoms, being present in about one-third of the cases. Nocturia was present in about 15 per cent. Other symptoms occasionally seen were incontinence, a sense of incomplete emptying of the bladder, pain in the bladder region and renal colic.

External irradiation has not proved as effective as was hoped. In some cases the results may be improved by the careful use of radon and radium therapy. Contact and possibly supervoltage therapy may prove valuable.—*Audrey G. Morgan.*

NERVOUS SYSTEM

Thompson, Ralph M. Neuroblastoma (sympathoblastoma or neurocytoma) of the suprarenal medulla. *Ann. Int. Med.*, June, 1942, *16*, 1206–1220.

There are three types of tumor derived from the sympathetic nervous system of the suprarenal medulla: neuroblastoma, ganglioneuroma and paraganglioma (chromaffin cell tumors). The author considers only the first type which occurs almost entirely in infants and young children and occasionally in young adults.

He describes 3 cases in patients seventeen months, five years and eighteen years of age. Photomicrographs of the findings are given. The younger the patient, the less differentiated the cells in these tumors and the greater the malignancy. The patients generally grow progressively worse and die. In these cases the periods of hospitalization were 40, 191 and 105 days respectively.

Pepper has claimed that these tumors metastasize only to the liver and Hutchinson that they metastasize to the bones, chiefly those of the skull. The clinical symptoms are the same in the two groups. Frew claims that tumors of the left suprarenal metastasize to bones through the blood and those of the right suprarenal to the liver through the lymphatics.

The diagnosis depends largely on the metastatic lesions and the presence of an abdominal tumor.

Contrary to generally accepted opinion, these tumors are radiosensitive and life may be prolonged by irradiation. In the second case of this series a total dose of 4,500 r was given and the abdominal tumor shrank from the size of a child's head to a nodule 3×1.5×1.5 cm. in size found on autopsy. There is no doubt that life was prolonged by the irradiation but autopsy showed marked aplasia or destruction of bone marrow.

One case has been reported in which a patient was alive fifteen years after operation but in general surgery is not successful in these cases.—*Audrey G. Morgan.*

Hauser, Emil D. W. Neurofibroma (neurinoma) of the foot. *J. Am. M. Ass.*, April 10, 1943, *121*, 1217–1219.

Hauser reports the case of an eleven year old girl who had pain in the ball of the right foot for six months. Roentgenograms did not aid in diagnosis. At operation a neurinoma (neurofibroma) which was found attached to the plantar fascia was removed. Six months later the pain recurred. At the second operation a large sausage-shaped neurinoma (neurofibroma) was found beneath the plantar fascia and removed. The tumor was easily dissected from the tendon sheaths in the area and was traced up until it ended in the deep plantar nerve.

Ewing states that this type of tumor affects mainly the larger nerve trunks and occurs in nearly all parts of the body, forming encapsulated, slowly growing, firm or soft masses attached to the nerve trunk. They have a definite tendency to recur locally from remaining tumor tissue if not completely removed and may become malignant and locally invasive following recurrence.—*S. G. Henderson.*

SKELETAL SYSTEM

Kleinberg, Samuel, and Burman, M. S. Spondylolisthesis; report of 3 cases in adults with forward displacement of the vertebra below the level of the laminar defect. *J. Bone & Joint Surg.*, Oct., 1942, *24*, 899–906.

The authors state that it has been the consensus that the essential factor or *sine qua non* in spondylolisthesis is a bilateral defect separating the posterior arch from the body of the vertebra. Moreover, according to all records, the slipping always occurred in the vertebra with the laminar defect. They report 3 cases which they consider unusual for the following reasons:

1. The vertebra which became displaced was not the one with the laminar defect but the one below it.

2. In 1 of the cases, the laminar defect was apparently unilateral, the subluxation occurring with the apophyseal joint of the uninjured side as the center of rotation.

3. In each instance, there was a trauma adequate to produce a fracture of the laminae; the symptoms appeared after the injury, and hence it seems possible that the slipping resulted from a laminar fracture—that is, that the spondylolisthesis was of traumatic origin.

4. Although the patients were adults, the slipping in each instance was only mild.

5. In 2 of the patients, the laminar defects appeared identical in location and extent, and there was a similar displacement and rotation of the posterior arches.

The authors remark that it remains for future experience to determine whether these findings necessarily indicate that the laminar defect is congenital, and militate against the suggestion that the spondylolisthesis is of traumatic origin. —*R. S. Bromer.*

LYON, ERNST. Intercalary bones of the intervertebral disc. *J. Bone & Joint Surg.*, Oct., 1942, *24*, 805–811.

Lyon defines the intercalary bone of the intervertebral disc as an osseous formation completely surrounded by the tissue of the intervertebral disc; that is, it is interposed between the two vertebral bodies. Intercalary bones are irregularly shaped, isolated osseous formations and are usually found in the anterior peripheral portion of the disc. They occur in older individuals who suffer from spondylosis and are found mainly in the discs of the lumbar region, although they are occasionally encountered in the thoracic and cervical portions of the spinal column.

On roentgenographic examination, the posteroanterior view reveals irregularly outlined, small or larger shadows in the intervertebral space, in the neighborhood of the vertebral corner. They are usually situated between two opposite spondylotic osteophytes. The lateral view shows them as homogeneous, triangular shadows, the size of a rice kernel or even larger, situated in the anterior portion between the upper and lower edges of two adjacent bodies. They sometimes touch the edge of the vertebral body and may approach two opposite spondylotic osteophytes. An open space usually appears between these shadows and the vertebral surface.

Considering the high incidence of spondylosis, these bones provide a rare accidental finding of only secondary importance. Symptoms, if any are present, are not characteristic of the condition. There are no particular symptoms definitely connected with the presence of the intercalary formations. They do not occur as the sequel to an accident and they represent the ultimate stage of a curative process in a lesion of the outer portion of the disc and of the nucleus pulposus, which has been present for a considerable time.—*R. S. Bromer.*

BAKER, LENOX D. Rhizomelic spondylosis; orthopaedic and roentgen therapy. *J. Bone & Joint Surg.*, Oct., 1942, *24*, 827–830.

Baker in this report describes the apparatus and routine used in 22 cases of rhizomelic spondylosis treated by roentgen therapy combined with well established orthopedic measures. The therapy was directed at alleviating pain, relaxing muscle spasm, and correcting deformities, with the hope of arresting the disease. Baker employed the principles outlined by Swaim which consisted of gradual hyperextension of the spine, relaxation of muscles, corrective exercises to straighten the spine and increase chest expansion, and immobilization of the spine to hold the correction. Swaim believed that the trauma of use plus continued muscle spasm served to keep the disease active, and to keep the involved tissues in an inflamed condition during convalescence. He used plaster of paris on leather body jackets to obtain complete immobilization which he did not believe was accomplished by the popular type of spinal braces.

Baker found that in a majority of patients severe pain was usually present during the early period of treatment. The addition of roentgen therapy gave an early alleviation of pain with relief of muscle spasm and it allowed rapid correction of deformities and more vigorous attempts at restoration of muscle balance. The roentgen therapy, if not contraindicated, was given over either the entire spine or the involved area, depending on the clinical findings. Two hundred kilovolts were used in treating areas of about 5 by 15 cm; 150 r was given over one or more areas daily for three to five treatments; if necessary, the dosage was repeated after three to six weeks.

The average length of hospitalization with the combined therapy was ten days. The cor-

rection of deformities, thus far, has been satisfactory. In the cases treated, follow-up roentgenograms showed no increased calcification in the spinal ligaments and the patients as a whole maintained the increase which had been obtained in the range of motion. The most satisfactory results were obtained in the early cases but surprisingly good improvement was noted in some of the patients in whom marked deformity and advanced calcification were present.—*R. S. Bromer.*

Hart, Vernon L. Primary genetic dysplasia of the hip with and without classical dislocation. *J. Bone & Joint Surg.*, 1942, *24*, 753–771.

Classical congenital dislocation of the hip is secondary to a primary genetic dysplasia of the hip, or flat acetabulum, and develops during intra-uterine life, or in the course of the first or second year of postnatal life. Dislocation is a consequence of the primary dysplasia or "flat socket"; it is a secondary and incidental phenomenon. It has been difficult to explain genetically congenital hip dislocation. An individual does not inherit the condition but does inherit a primary defect, or hip dysplasia, which produces anatomical and physiological alterations of the joint which is called the "flat socket." To make a true genetic investigation of hip dislocation, roentgenographic consanguinity studies are essential according to Hart's opinion. In his investigation he found it cannot be sex-linked because it is observed to pass from father to son. There is no "dislocation" gene but there is a "hip dysplasia" gene. The gene involved is not a recessive gene but is a dominant one.

Hart believes that dysplasia of the hip without dislocation is a distinct clinical and roentgenographic entity, and should be included in textbooks in the chapter on classical dislocation, because the two entities have the same genetic etiology. Dysplasia of the hip without dislocation occurs more frequently and may be more disabling than classical dislocation. Acclivity of the roof of the acetabulum is the primary anatomical feature of a dysplastic hip joint. Hip dysplasia refers not only to the acetabulum, but also to all joint-forming parts of the hip joint. Primary hip dysplasia can lead much more easily to complete dislocation in the female than in the male. The condition with dislocation on one side is frequently associated with dysplasia without dislocation on the opposite side. It may remain asymptomatic for many years; in some individuals it may never be shown clinically. The extreme disability observed in some individuals is caused by traumatic arthritis of the hip joint resulting from mechanical factors of instability and incongruity.

Hart emphasizes proper roentgenographic technique to prevent distorted views and erroneous measurements of the hip joint. The patient must be relaxed and placed flat on the back. The lower extremities should be in contact, the hips extended, and the patellae facing directly forward. The roentgen tube must be centered in the midline of the body and directly over the superior border of the symphysis pubis. Roentgenographic diagnosis of hip dysplasia without dislocation is very difficult during the first months of life, and the condition is rarely recognized except where the routine examinations of the newborn are made. Most often it is dislocation of the opposite hip which leads to its discovery.

The following hip joint lesions may at times require careful study for differentiation from primary hip dysplasia: coxa plana or Legg-Perthes' disease, epiphyseal separation, tuberculosis, infectious arthritis, hypertrophic arthritis and aseptic necrosis. To these should also be added: late septic hip joint, neoplasm, neurotrophic lesions, old fracture-dislocations, and endocrine dysfunction.

The article is well illustrated with roentgenograms and the treatment is described in detail. The patient may be relieved of pain and disability by conservative treatment and surgical measures.—*R. S. Bromer.*

Compere, Edward L., and Wallace, George. Etiology of aseptic necrosis of the head of the femur after transcervical fracture. *J. Bone & Joint Surg.*, Oct., 1942, *24*, 831–841.

The incidence of non-union resulting from treatment of intracapsular fractures of the hip by methods of internal fixation has been found to be very high. The primary etiological factor in aseptic necrosis is disruption of the blood supply to the head of the femur. The development of techniques for obtaining satisfactory lateral roentgenograms of the hip has undoubtedly resulted in more accurate reduction of transcervical fractures. The authors planned an experiment to try to determine whether or not immediate reduction and complete immobilization by internal fixation of the fragments would

influence favorably the incidence of aseptic necrosis of the head of the femur.

In the experiments carried out, necrosis with collapse of the weight-bearing portion of the cortex of the head occurred in 2 of 6 cases following accurate reduction of the fracture and pinning of the fragments, although the fracture united. In these 2 cases the head of the femur may have been viable when union occurred but, with partially depleted blood supply, the minimal traumata of continued use and weight bearing may have resulted in the necrosis and disintegration.

Compere and Wallace conclude that necrosis of the head of the femur occurs less often if the fracture of the transcervical neck is immediately and accurately reduced and adequately immobilized. The prognosis for survival of the head of the femur is definitely less satisfactory if the reduction of the fracture is not anatomically correct or if pinning of the fragments is not adequate for complete immobilization.

They found that death of the head of the femur when the fracture was neither reduced nor immobilized occurred in 9 of the 12 hips studied. It is possible, they believe, that weight-bearing activity and friction between the fragments may have ruptured the blood vessels that were left intact after fracture in a portion of the posterior capsule. However, this theory does not explain the difference in incidence of necrosis in cases in which the head was pinned without accurate reduction and in those in which the head was pinned in an anatomically correct position.

The union of a fracture of the neck of the femur, regardless of the method of reduction or of immobilization, does not constitute of itself an end-result. The condition of each head of the femur should be studied at intervals for at least five years after union and beginning weight bearing by those surgeons who are qualified by training and experience to recognize roentgenographic changes and to interpret them in terms of pathological processes such as aseptic necrosis.—*R. S. Bromer.*

Hefke, Hans W., and Turner, Vernon C. The obturator sign as the earliest roentgenographic sign in the diagnosis of septic arthritis and tuberculosis of the hip. *J. Bone & Joint Surg.*, Oct., 1942, *24*, 857–869.

In the anteroposterior roentgenographic examination of the pelvis and hip, a soft tissue shadow may be seen just medial to the acetabulum on the inner aspect of the pelvis. The border of this shadow ordinarily starts from a point lateral to and below the lower pole of the sacroiliac joint, follows the line of the pelvic inlet downward over the acetabulum, and disappears behind the upper ramus of the pubis. This shadow in normal children measures from 2 to 8 mm. at its widest point. Usually the shadow on one side corresponds in width to the shadow on the other. There may be, however, a minimal difference in width of this shadow in normal individuals. It has been noted that certain changes occur in the contour of this shadow in cases of septic arthritis and in tuberculosis of the hip. The authors' purpose in their study reported in this paper was to determine the anatomical basis of this shadow and its importance in the early roentgenographic diagnosis of pathological conditions about the hip.

Hefke and Turner believe that the obturator internus muscle has been definitely proved to be the substratum of the shadow. The fibers of the obturator internus were laid bare in a postmortem specimen, and a wire was placed along its medial border. This corresponded exactly to the position of the border of the shadow as seen in all the roentgenograms. In regard to the pathological changes underlying the variation of the shadow in septic arthritis, the tendon of the obturator internus passes immediately adjacent to the capsule of the hip, and extension of the inflammatory process may well lead to a roentgenographically visible swelling of the obturator muscle.

In a small number of cases it was noted that the obturator shadow was obscured. After making the study the conclusion was reached that an obscured obturator shadow is a positive finding indicative of pathological changes in the hip joint. They also believe the widening, the changes in contour, and the obscurity of the border, may be compared with the well known changes of the psoas shadow seen in roentgenographic examination of a psoas abscess, perinephritic abscess, or other inflammatory process overlying, or in contact with, the psoas muscle.

They advise the use of the usual technique for routine roentgenograms of the pelvis. No special advantage was shown in the use of soft tissue technique. Care must be taken that the pelvis is level or one obturator may be thrown into higher relief than the other one. They found that in some cases it was advantageous to obtain an anteroposterior view of the pelvis with the hips in marked abduction, flexion to 90

degrees, and marked external rotation (so-called "frog position," frequently used in obtaining lateral views of the hip joint) which seemed to bring the obturator shadow into greater relief.

They conclude that in certain types of pathological conditions of the hip joint the obturator shadow becomes obscured, or it becomes widened with a more curved border. The greatest value of the sign is in early roentgenographic diagnosis of septic hip disease. It is of some importance in early diagnosis of tuberculosis of the hip. It was not present in fractures about the hip, osteochondritis, slipping of the capital epiphysis, rheumatic fever with clinical involvement of the hip, osteomyelitis of the pelvis not involving or neighboring the hip joint and soft tissue infection about the thigh with marked inguinal lymphadenopathy.—*R. S. Bromer.*

Weaver, James B. Calcification and ossification of the menisci. *J. Bone & Joint Surg.*, Oct., 1942, *24*, 873–882.

Weaver reports 2 cases of calcification of the menisci of the knee joint. In addition to his 2 cases, he found reports of 76 others in the literature. Primary or non-traumatic and secondary or traumatic calcifications are two separate and distinct entities that have little in common except the presence of calcium. The primary type occurs in older individuals, the secondary in younger. There was a preponderance of males in the series of cases in both types. Both menisci of both knees were affected in cases of the primary type, but only one meniscus in each knee was affected in cases of the secondary type.

In the primary cases, the calcium is laid down in parallel layers starting at the periphery of the cartilage and involving the entire length. The meniscus is smooth. The secondary cases show a localized zone of calcification, usually in one end or the other, and of the central type. This calcium piles up, causing a roughening and enlargement of the part of the cartilage affected.

In the roentgenogram, the primary type shows a waferlike aspect of the cartilage in the anteroposterior view and wedge-shaped shadows at each end of the meniscus in the lateral view. This suggests a diffuse calcification of all four menisci, although these areas may vary in size and density according to the age of the lesion. Calcification may be revealed in individual menisci at various times in the same patient. The roentgenographic appearance is characteristic and diagnostic. The secondary type shows a localized piling up of calcium, rough in outline, which might be confused with a joint mouse or ossification in the cartilage. The picture is not diagnostic.

In many instances primary calcification produces no symptoms. In some cases, symptoms such as pain, swelling, limitation of motion and occasionally hydrops, are severe enough to necessitate removal of the cartilage. Pain, swelling and limitation of motion are the principal symptoms of the secondary type. Hypertrophic arthritis, both in the affected joints and in other joints, was almost a constant finding in the primary cases.

Weaver states that in the primary type, the treatment is symptomatic. Excision of cartilage is infrequently necessary. Spontaneous cures were numerous in the reports in the literature. Extirpation is always necessary in the secondary type.

Ossification of the semilunar cartilage is a condition closely allied to calcification of the cartilage and may be a sequela to calcification. The symptoms, roentgenographic appearance, and treatment of secondary ossification are practically the same as in secondary calcification. Weaver found no evidence that primary calcification is a precursor of secondary ossification. He is of the opinion that many cases of secondary calcification do not progress to ossification. The cases of secondary ossification reported by Wollenberg, Eck, and Weaver are unquestionably true examples of heterotopic bone formation, and the other cases reported are probably in the same category.—*R. S. Bromer.*

Freud, Paul, and Slobody, Lawrence B. Symphalangism; a familial malformation. *Am. J. Dis. Child.*, April, 1943, *65*, 550–557.

Symphalangism results from failure of an interphalangeal joint to form. There may be complete or partial lack of development of the joint. When the interphalangeal joint is entirely absent, there is no dividing line between the two phalanges. They appear, rather, as a single long phalanx with a common bone marrow cavity. The trabecular structure of the bones is changed to conform to the new stresses of pull and pressure. When the interphalangeal joint is developed slightly, there are two distinct bones and two bone marrow cavities. The ligaments are shorter than normal and there is a solid fibrous connection between the two bones. Motion is impossible. When the development of the interphalangeal joint has progressed a little further

some bending and stretching become possible.

In the family reported in this paper, the family tree covered four generations. Nine of 10 members of the family were known to have symphalangism. This illustrates the dominant character of the inherited malformation. The roentgenograms which are reproduced in the paper illustrate varying degrees of the condition and demonstrate the manner in which the various stages of articular differentiation occur during the life of the embryo. The earliest change is a constriction of the phalangeal beam. This progresses until only a central bony bridge remains. This bridging becomes less distinct and then invisible, and then a normal joint space is formed. The process may be stopped at any point before complete differentiation with resulting symphalangism.—*R. S. Bromer.*

STONE, SAMUEL. Osteomyelitis of the long bones in the newborn. *Am. J. Dis. Child.*, Oct., 1942, *64*, 680–688.

There are few reports of osteomyelitis of the long bones in the newborn. Osteomyelitis in the newborn child appears to be a benign disease; but when it is a manifestation of sepsis, the prognosis is more grave and depends on the severity of the septic process. Stone reports 4 cases in which the striking feature was the benign course of the disease. Characteristic were the quick healing of the osseous lesions, as observed in the roentgenograms, the rapidity of subperiosteal new bone formation, the short duration of discharge from sinuses and the quick healing of the operative wounds. In none of his patients did sequestration occur. None of his infants was acutely ill, and the admission to the hospital was usually sought because of localized swellings of the extremities or diminished motion of the arms and legs rather than because of severe systemic symptoms. The general condition of the infants remained good; they took their feedings well and gained weight. The operative procedures were limited to incision and drainage of palpable localized abscesses without involving the bone. Green and Shannon ascribed the benign course of osteomyelitis in infants to the peculiar anatomy of the bone at this age. According to them, the vascular spaces of the bone are larger and the bone is of spongy texture and less rigid than in older children. The thin cortical bone at the metaphysis allows freer communication between the marrow and the subperiosteal spaces and thus provides a mechanism for decompression of pus from the metaphysis and the marrow to the subperiosteal spaces. The periosteum is more loosely attached in infants, and this further aids decompression. Rupture of the periosteum, with decompression of pus, also makes for absence of gross sequestration. The problem in treatment, as with osteomyelitis in older children, is to tide the patient over the period of bacteriemia. The treatment of the osseous lesion is of secondary importance.—*R. S. Bromer*

BARBER, C. GLENN. Osteochondrosis deformans tibiae; nonrachitic bow leg in children. *Am. J. Dis. Child.*, Nov., 1942, *64*, 831–842.

Barber states that at the time of writing the most common cause of development of bow leg during childhood in his community is osteochondrosis of the medial tibial condyle. He believes that the reason the condition has not been more widely recognized is due to the fact that it has been described by a diversity of names in previous reports. Since Blount called attention to it as a cause of acute bending of the legs in children, he has encountered no less than a dozen cases in which the deformity might be rightfully attributed to osteochondrotic changes in the tibial epiphyses. Although the children had received adequate amounts of vitamin D and although other evidence of rickets was wanting, the parents had either been told or were of the opinion that the deformities of the legs were due to that disease.

The roentgenographic and pathologic changes are like those of coxa plana and those in the many bones similarly involved elsewhere but are quite different from those of rickets. When the tibial condyles are involved, two distinct types occur. These two types are dependent on the age at which the condition becomes manifest, and they have been designated as the infantile and adolescent types of the disease denominated by the less cumbersome though more grossly descriptive term "tibia vara."

In cases of infantile type a history of normal development, except for some overweight, for one or two years is usually obtained. The deformity occurs bilaterally and when mild may disappear on one or both sides spontaneously. When it is unilateral there is a limp in walking, and when it is bilateral there is a waddle. When the deformity occurs during infancy, a bulbous enlargement of the medial tibial condyle is palpable on physical examination and visible in the

roentgenogram. This beaklike projection at the metaphysis has been shown to contain islands of cartilage. Faulty growth of the upper tibial epiphyseal cartilage and delayed ossification of its medial half result in a wedge-shaped deformity of the epiphysis. The lower tibial epiphyseal cartilage occasionally shows similar though less pronounced changes; if this occurs, some lateral bowing just above the ankle may appear. Elsewhere the tibial shaft is perfectly straight.

The onset of the adolescent type is between the ages of six and twelve years in previously normal children. The deformity usually occurs on one side only. The adolescent type looks different in the roentgenogram. It is an arrest of growth rather than a dysplasia. The roentgenographic appearance of the infantile type gradually changes to that of the adolescent type, so that the two can be distinguished later only by the history. Recurvation at the knee and relative flatfoot are present, irrespective of the age.

The treatment of osteochondrosis deformans tibiae depends on the stage at which it is instituted. Measures to prevent further deformity or to correct existing deformity, even when extreme, have proved effective during the plastic stage of the condition. When deformity is disabling or unsightly after the osteochondrosis has become arrested, tibial osteotomy is advisable. However, if it is performed before arrest is assured, recurrence of the deformity is to be expected.—*R. S. Bromer.*

Brakeley, Elizabeth. Late rickets. *Am. J. Dis. Child.*, Feb., 1943, *65*, 314–319.

A case of late rickets in a girl aged eleven is reported. Active rickets after two years of age is uncommon, and only a few cases have been reported of its occurrence in older children who have been extremely resistant to treatment and who required massive doses of vitamin D before cure was accomplished. In the case reported in this article, healing began after three months, following a daily dose of 40,000 U. S. P. units of vitamin D. The child continued to show extreme decalcification of bones, a high serum calcium level, a low level of serum phosphorus and a high serum phosphatase level for fifteen months after healing of the rickets. It was believed that she had a fairly high degree of hyperparathyroidism secondary to the rickets, although no parathyroid glands were found on operation. Gradual improvement followed irradiation of the neck in the region of the parathyroid glands. The dosage given at the first treatment was 800 r to the right side of the neck, followed by the same dosage twenty-three days later to the left side of the neck. It is impossible to prove, however, whether the gradual increase in the calcification of the shafts of the bones and the return to normal values of serum calcium, phosphorus and phosphatase were a result of the irradiation or would have occurred without further treatment.—*R. S. Bromer.*

BLOOD AND LYMPH SYSTEM

Chamberlain, Francis L. Diseases of the lesser circulation. *Radiology*, August, 1942, *39*, 151–152.

Regulation of the lesser circulation differs from that of the greater circulation and these differences must be known in order to understand diseases of the lesser circulation. The most important abnormal condition in the lesser circulation is hypertension, which is most frequently caused by failure of the left ventricle. This causes engorgement from backing up of the blood in the pulmonary circuit. If the right ventricle fails at the same time this engorgement does not occur. Other less frequent causes of pulmonary hypertension or engorgement are mitral stenosis, and congenital, traumatic or syphilitic left-to-right heart shunts.

Pulmonary hypertension is usually associated with pulmonary engorgement because of the large diameter of the pulmonary capillaries. This explains the variability in the roentgenogram of the lung in the common types of heart disease. It also explains why enlargement of the heart must take place before pulmonary hypertension with its attendant dyspnea and other symptoms can occur. It explains the increased density and size of the hilar shadows, increased lung markings and the radiopaque appearance of the lung fields proper.

Diseases of the lesser circulation characterized by intrinsic or extrinsic obstruction of the pulmonary vessels (cor pulmonale) may be acute, subacute or chronic. The clinical signs may differ from those of pulmonary hypertension due to the relative absence of pulmonary engorgement. There may be no râles and the lung fields may be unusually radiolucent. But underlying lung disease may confuse the picture. Cyanosis is usually more marked and in the chronic types the fingers and toes may be-

come clubbed and polycythemia may develop. —*Audrey G. Morgan.*

STEINBERG, M. F., GRISHMAN, A., and SUSSMAN, M. L. The angiocardiographic demonstration of an arteriovenous fistula. *Surg., Gynec. & Obst.*, July, 1942, *75*, 93–96.

The roentgen demonstration of peripheral arteriovenous fistulas has been accomplished by the introduction of radiopaque substances through direct arterial punctures. A simpler method is available through the application of the angiocardiographic technique. This consists of the rapid introduction of diodrast 70 per cent through a special needle into an antecubital vein, making serial roentgen exposures which may be recorded either fluorographically or roentgenographically.

A case is described in which the clinical picture was unmistakably that of an arteriovenous fistula of the subclavian or axillary vessels. However, more accurate localization of its site was considered desirable before operative intervention because the surgical approach for a fistula between the subclavian vessels would be transthoracic while that for a fisutla between the axillary vessels would be entirely extrathoracic. Two injections of 30 cc. of diodrast 70 per cent were given; the first into the left antecubital vein, the second into the right antecubital vein. Roentgenograms were taken rapidly with the roentgen tube centered over the supraclavicular region, shoulder, and upper arm. Injection of the involved side definitely established the identity and size of the left axillary, subclavian and innominate veins and the axillary artery. It was evident that the opaque material must have reached the axillary vein directly from the axillary artery at a point near their origins, since both of these vessels were simultaneously visualized. It was likely that the actual fistula was present at a point proximal to the origin of the axillary vessels. The third portion of the subclavian artery was therefore designated as the probable site of the fistula.—*Mary Frances Vastine.*

WATSON, JAMES R., LICHTY, J. M., HILL, J. M., and MILLER, R. B. The use of venograms for the localization and study of arteriovenous fistula. *Surg., Gynec. & Obst.*, June, 1943, *76*, 659–664.

Venograms were performed on 3 patients with arteriovenous fistulas. These cases are reported in the article.

In traumatic arteriovenous fistula, the changes in the vein consist of hypertrophy and dilatation. The dilatation may be diffuse and extend for some distance proximal to the level of the fistula or it may be maximal locally at the site of the fistula. As long as the valves remain competent, no changes are apt to occur in the venous trunk distal to the fistula. Dilatation in the distal portion is uncommon for the arterial blood, taking the course of least resistance, passes through the fistula and into the proximal vein. The essentially normal status of the involved vein distal to the level of the fistula makes it readily adaptable for venography.

Indications for Venography.

1. Demonstration of fistulas at or proximal to the roots of the extremities where injection of the artery (arteriogram) above the level of the fistula would be difficult or even impossible because of its anatomical position.

2. Venography might be used in conjunction with arteriography when the presence of multiple fistulas is suspected.

3. In cases in which arteriography or venography could be used, venography has the advantage of permitting the use of media which are inert when injected into the venous circulation and which are readily excreted by the kidneys.

In their summary, the authors include the following statements:

1. Studies of the venograms in each instance showed the vein distal to the fistula to be normal.

2. The venograms demonstrated a marked difference in the collateral venous circulation between 1 case in which the vein had been ligated distal to the fistula at the time of injury for control of hemorrhage and 2 cases in which this had not been necessary.

3. Ligation of the vein distal to the fistula appears to have the same beneficial effect on the extremity that ligation proximal to the fistula has on the heart.—*Mary Frances Vastine.*

ROENTGEN AND RADIUM THERAPY

WEED, L. A., ECHTERNACHT, A. P., MEISTER, E. J., and ISENHOUR, ROGER. The effect of therapeutic doses of x-ray on infections and inflammations; experimental studies. *Surg., Gynec. & Obst.*, Aug., 1942, *75*, 157–160.

Part of the data for this paper was obtained at the State University of Iowa and the remainder at the Indiana University Medical Center. The summary and conclusions reached are:

(1) In guinea pigs roentgen irradiation had no beneficial effect in the treatment of gas gangrene due to *Clostridium welchii*.

(2) Roentgen irradiation was ineffective in reducing the number of viable vegetative forms of a two hour culture of *Clostridium welchii*.

(3) Roentgen irradiation was ineffective in retarding the rate of growth of *Clostridium welchii*.

(4) Roentgen irradiation did not prevent the development of hemolysin or dermonecrotic toxin of *Clostridium welchii*.

(5) Roentgen irradiation would not prevent the development of dermonecrosis even if used before or immediately after the injection of *Clostridium welchii* toxin.

(6) Roentgen irradiation would not inactivate either the hemolysin or dermonecrotic toxin when applied in vitro.

(7) Roentgen irradiation would not inactivate in vitro either the hemolysin or dermonecrotic toxin of *Staphylococcus aureus*.

(8) Roentgen irradiation would not inactivate in vitro the toxin of *Corynebacterium diphtheriae*.

In no experiment has any real effect been demonstrated with doses of roentgen rays even beyond that permitted for human therapy.—*Mary Frances Vastine.*

Medical Uses of Radium; summary of reports from experimental research centers for 1940. Edited by the Joint Radiology Committee of the Medical Research Council and the British Empire Cancer Campaign. *Brit. J. Radiol.*, Feb., 1942, *15*, 56–62.

This is a continuation of the series of articles on Medical Uses of Radium which has been issued by the Medical Research Council for many years. It describes the work of the Radium Beam Therapy Research Team in London, including a study of roentgen-ray and gamma-ray effects. The construction of the pressure-insulated two-million volt electrostatic generator is proceeding satisfactorily and further experiments have been made on the properties of moulded bakelite-graphite mixtures to which traces of heavy elements have been added. A mixture containing calcium fluoride has proved better than the ones previously used. The Royal Cancer Hospital in London reports work on the spatial distribution of radiation during radium treatment. The Mount Vernon Hospital of Northwood reports a method of measuring very small partial discharges of a condenser dosimeter. The Strangeways Research Laboratory in Cambridge reports generalizations on the biological actions of radiation, the inactivation of plant viruses by radiation, the production of chromosome abnormalities in irradiated plant material, the effect of gamma rays on normal embryonic cells *in vivo* and the quantitative biological analysis of human biopsies. The Department of Medicine of the University of Cambridge reports on the measurement of the effects of roentgen and gamma irradiation on normal and malignant cells by means of ultraviolet photomicrography. Dr. Gray and Dr. Read of the Mount Vernon Hospital, Northwood, report on the biological effects of ionizing radiations, the summation of neutron and gamma-ray effects, the neutron irradiation of mouse tumors *in vivo* and *in vitro* and the lethal effect of alpha radiation on bean roots. The Barnato Joel Laboratories of the Middlesex Hospital, London, report on the effects of irradiation on rabbits' ovaries, using radon instead of radium.—*Audrey G. Morgan.*

Wilson, C. W. Method of "dose-finding" for combinations of rectangular radiation fields. *Brit. J. Radiol.*, May, 1942, *15*, 145–149.

The methods of dose-finding heretofore in use have applied only to circular fields. In this article a method of representing the three-dimensional distribution of radiation throughout rectangular radiation fields is described. For each roentgen-ray field the dose contours are determined by measurements in a pressdwood phantom in a number of planes containing the central ray, at various angles to one of the major axes of the field. The measurements are made with a Siemens dosage-rate meter. The apparatus is illustrated and dose contours in various planes are given.—*Audrey G. Morgan.*

Levitt, W. M. Discussion of the constitutional effects of radiation, with special reference to volume dose. *Brit. J. Radiol.*, April, 1942, *15*, 99–103.

This was a discussion by the Faculty of Radiologists with Dr. Levitt presiding as chairman. He discussed the term "volume dose" which means the relationship between the total amount of radiation energy absorbed and the constitutional effects produced. A "dose" of radiation does not mean a definite quantity. A dose may be given that has such serious constitutional effects that it more than nullifies the good local effect. Volume dose cannot always be calculated accurately but progress has been made in this respect. Even if the volume dose

is accurately calculated there are other circumstances to be taken into consideration, such as the effect on the blood-forming tissues and the decreased toleration to radiation in liver disease.

Dr. Levitt then introduced Prof. Mayneord who discussed the relationship between the roentgen and energy absorption per unit mass of air and tissues. He recommended the use of the megagram-roentgen as a unit, as the gram-roentgen is far too small. The megagram-roentgen is the energy absorption if a uniform dose of 1,000 roentgens were given throughout 1,000 grams of air or soft tissues. He also discussed the energy absorption in the beam for different techniques and the relationship of total energy absorption to protection and its dependence on wave length. The chemical effects of roentgen rays were discussed and a method of calculation of optimum penetration given.—*Audrey G. Morgan.*

SMITHERS, D. W., ELLIS, FRANK, and GRIMMETT, L. G. Discussion on the constitutional effects of radiation, with special reference to volume dose. *Brit. J. Radiol.*, May, 1942, *15*, 141–144.

Dr. Smithers in a meeting of the Faculty of Radiologists discussed the complexity of the task of measuring the total amount of radiation energy absorbed by the body. The clinical problem is much more complex than the physical one and even the physicists have had difficulty in measuring integral dosage. The integral dose, measured in megagram-roentgens, is a far more valuable index of the treatment given than the surface dose. He gives a table showing the integral doses and the tumor doses in two groups of cancers, one of the pharynx and larynx and one of the stomach and esophagus; the tumor doses were the same in the two groups but the integral doses were more than twice as large in one group as in the other.

He also discussed the use of vitamin C in association with irradiation and the apparent good results of such treatment, and the use of histaminase in the treatment of roentgen sickness.

Dr. Ellis said that the volume dose of total energy absorbed is given by the product of the area of the field, the surface dose, a graph reading and the ratio of the time to deliver the dose to the field concerned, divided by that to deliver the same dose to a field at the center of which the scattering is maximal. However, there are inaccuracies in this and all other methods and an accurate estimation of total energy absorbed is as yet impossible.

It has been found that the fatal volume dose per kilogram in a rat is about the same as that of a single dose of roentgen rays to the whole human body. This suggests that some toxic substance may be produced by irradiation that has the same quantitative action on man and animals.

Dr. Grimmett described a method of measuring the total energy absorbed by the human body by the use of a life-size celluloid man in which the sum of the ionization currents in the air-gaps between the celluloid plates is measured. It offers a quick method of measuring energy absorption for gamma rays and high voltage roentgen rays.—*Audrey G. Morgan.*

APTHOMAS, INEZ. Contact x-ray treatment of cavernous angioma in children. *Brit. J. Radiol.*, Feb., 1942, *15*, 43–46.

Mention is made of an article in the AMERICAN JOURNAL OF ROENTGENOLOGY AND RADIUM THERAPY, November, 1939, which reviews cases of the above-named disease treated from 1933–1936. This article brings the record of the treated cases up to date and describes a new method of treatment used in the Holt Radium Institute, Manchester, England, since 1936. The new method is that of contact roentgen therapy, using a Chaoul tube at 60 kv. and 4 ma. The applicators range from 1.5 cm. in diameter at 15 cm. focal skin distance to 9 cm. in diameter at 10 cm. focal skin distance. The smaller ones are usually adequate.

This treatment is used only in cavernous angioma proper and solid hemangioma. The capillary type, or port-wine stain, is almost completely radioresistant. For the average lesion a dose of 400 r is sufficient, repeated at intervals of two months. Tables are given showing the results for the radium methods used before 1937 and the contact roentgen therapy cases treated since then. There were about 92 per cent cures for both methods, the other 8 per cent being improved. The contact therapy takes less time than the elastoplast method; no anesthetic is needed and it is never necessary to remove an unsightly gold seed. Radium implants are to be preferred in very bulky lesions. The number of treatments required varies with the dose and the size of the lesion.—*Audrey G. Morgan.*

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VOLUME 51 JUNE, 1944 NUMBER 6

Medical Library JUL 5 1944

[FOUNDED IN 1906 AS THE AMERICAN QUARTERLY OF ROENTGENOLOGY]

The AMERICAN JOURNAL OF ROENTGENOLOGY AND RADIUM THERAPY

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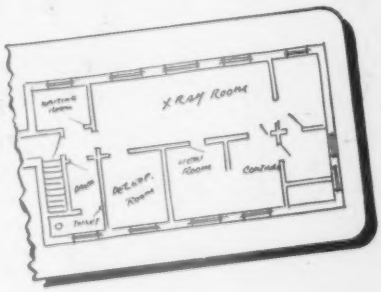

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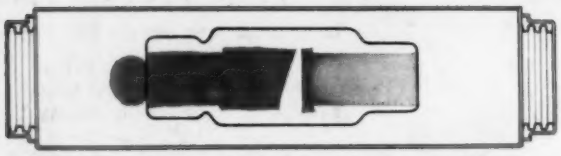

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